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ORIGINAL MEMOIRS.

ACUTE HÆMATOGENOUS INFECTION OF THE KIDNEY.

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THAT the kidney may become infected by way of the blood stream is a fact long known, but it remained for Brewer¹ to bring to our attention a curious class of cases in which the hæmatogenous kidney infection does not imply a general pyæmic process, and to show that in certain of these infarcted infected kidneys there was no other gross septic lesion, and that the affected kidney was within reach of effective surgical attack.

With the appearance of his article, seven years ago, the infarcted septic kidney² resigned its pathological position as an exhibit, and "went surgical."

A new symptom complex, representing a definite lesion, open to attack, was presented to the surgeons.

I fear we have not taken full advantage of our opportunity. In 1908 Cobb presented an excellent series of eight cases, operated on at the Massachusetts General Hospital by

¹ G. E. Brewer: ANNALS OF SURGERY, 1904, xl, 1010.

² With all respect for Brewer, it seems to me that his "acute hæmatogenous infection" is a rather formidable title for daily use. May I suggest *septic infarcted*, or *infected infarcted*, kidney as a handier handle?

himself and by certain of his colleagues, and reviewed the previous published data,³ but since then the reports have been few and scattering. If we may judge by the published data of *demonstrated* cases in the hands of a few men, these cases cannot be very unusual.⁴ They must have occurred in the practice of all of us. I confess that I have recognized no cases previous to those here reported, but as I look back I have an uneasy suspicion (amounting almost to a certainty in one case that went on to the formation of a perinephritic abscess) that I have seen such cases before, and have failed to recognize them.

Believing that I am not alone in this matter, I present the two cases here reported, intrinsically not especially notable except perhaps for the happy outcome.

They are presented for three reasons:

1. All such cases should be reported until all surgeons are on the lookout for them.

2. These cases suggest a connection between kidney prolapse and the choice (if we may so phrase it) of the localization of the lesion, that seems not to have been noted previously.

3. They are examples of what may be done by *conservative* operation if cases are operated on early enough.

CASE I.—Mrs. E. L., age thirty-nine years, married, multipara, entered Nov. 9, 1910, sent to the hospital by Dr. J. J. Fitzpatrick of Charlestown, Mass., who writes that he saw her Nov. 5, 1910. She then gave a history of an injury to her right side two years ago, the result of a fall, at a time when she was six months pregnant. There was no obvious interference with the following childbirth.

When he first saw her, she complained of irritation of the bladder and of pain in the lower abdomen and about the urethra.

Nov. 8, she complained of pain, and examination showed tenderness below the edge of the liver and over the right kidney,

³Farrar Cobb: *ANNALS OF SURGERY*, 1908, xlviii, 680.

⁴Brewer, in his second article (*Surgery, Gynecology, and Obstetrics*, 1906, ii, 485), was able to cite 13 cases, personally observed.

and there was complaint of pain also in the back, about the kidney.

Nov. 9, at 6 P.M., her temperature rose to 103° F., the pulse to 100, and she complained of chilliness and sweating, and showed marked soreness in the right flank. The urine showed a specific gravity of 1018, and albumin was present. There was diarrhoea with moderate vomiting.

Dr. Fitzpatrick referred her to the City Hospital, with a diagnosis of acute nephritis, and she came to me with the admitting physician's diagnosis of appendicitis.

I saw her as an "emergency case" in the evening of Nov. 9, 1910. Physical examination was negative, except for moderate spasm of the abdomen as a whole, and tenderness over the right iliac region and in the right loin (costovertebral). The tenderness was most obvious over the appendix region, and the tenderness behind was not more than one often finds with a high appendix. The temperature was 103°, the pulse 120.

Vaginal examination showed nothing significant. There was a decided trace of albumin in the urine.

At the time I was not able to exclude an appendicitis, and consequently the first incision was made in front. Nothing abnormal was found save that the right kidney, obviously prolapsed, seemed larger than normal. The patient was turned over, and the conventional posterior kidney incision was made, and the kidney brought into view. Several whitish patches appeared, conspicuous against the background of a purple, congested, enlarged kidney. The capsule was split and reflected to either side, and a number of whitish areas stood out, dull white, outlined in purplish-black against a purple kidney. There was no pus. The white areas were obviously of embolic origin. The capsule was freely rolled back, and the infarcts cut into freely.

There seemed no reason for sacrificing the kidney, or for cutting deeper.

The rolled-back capsule was caught up (Edebohls's technic) and sutured to the muscle layers. A "cigarette" drain was left in.

There was no post-operative shock or other trouble, and four days later the records showed a temperature already fallen to normal, with a pulse of 86. The drain was still in, but there was no pus. The urine showed no albumin.

The drainage was removed on the ninth day. At no time was there any pus discharge.

Eleven days after operation the patient was "up and about," and the wound had healed solidly.

At this time the urine was of high color, slightly cloudy, specific gravity 1020, acid, very slight trace of albumin, no sugar. The sediment showed one coarse granular cast, some fine granular casts, some hyaline casts, epithelial cells (squamous only), and a few "compound granule cells."

The patient was discharged fourteen days after operation in good condition.

December 11, 1910, she entered the hospital on the gynæcological service to be treated for an incomplete spontaneous abortion. No note is made on the records of the service of any urinary complaint or renal complication, and no urine examination is recorded.

I did not see her again until February 14, 1911. At this time she had a fistulous track in the wound, which she said had developed in December. This later cleared up without any operation. At this time the urine showed no trace of albumin, and the sediment showed no renal elements. The kidney was firmly anchored, and was not abnormally tender. August 4, 1911, she reported in writing that she was well and in good condition.

CASE II.—Mrs. N. M., age thirty-four years, widow; multipara; previous health fair, though she had been overworked. For about three weeks previous to admission to the hospital she had been feeling "out of sorts," though up to a week before admission she had no definite symptoms.

During the week previous to her entrance to the hospital she had been "miserable," with abdominal pain (not sharply localized) and she had a good deal of nausea. For two days she had had sharp pain in the right lower quadrant of the abdomen, with vomiting, and had been unable to work.

The day before she entered the hospital she vomited repeatedly, suffered from general abdominal pain and tenderness, and had to have recourse to injections to move the bowels.

Her physician, Dr. T. P. Fitzgerald, of Brighton, Mass., sent her to the Boston City Hospital on December 8, 1910, on which date I first saw her. She then showed some tender-

ness of the abdomen, with involuntary spasm of the muscles of the right lower quadrant. Deep palpation with both hands showed a mass, obviously the enlarged and prolapsed right kidney, which was abnormally sensitive to pressure.

At this time the temperature was 99.8° , the pulse was 90. The white blood count showed 12,000.

The urine was turbid, of acid reaction. Albumin was present to a total between 0.125 and 0.25 per cent. There was no sugar present. No casts were found.

On December 10 the temperature was registered 98.8° , the white count 10,000. Nevertheless the pain persisted, rather more localized toward the right groin. My examination on this date showed well-marked tenderness of the right lower quadrant of the abdomen. The costovertebral tenderness in this case was present, but was not very well marked.

All things considered, however, it seemed clear that we were dealing with a renal, not with an intraperitoneal, condition, and when an operation was decided on (December 10) the loin incision was used.

The kidney was readily found, but was found in a position of well-defined prolapse. It was brought out into the wound, and showed greatly increased intracapsular tension. There were mottled whitish areas at the lower pole, but no clean-cut infarcted patches. The kidney was cyanotic, swollen, and was markedly prolapsed. The capsule was split, was rolled back, and was sutured to the muscles as in the previous case.

December 22 (the twelfth day), the temperature and pulse had fallen to normal and no albumin was present in the urine. No pain. Wound healed solidly. "Up and about" for the past four days. Discharged from the hospital.

The urine in this case showed *after* the operation: specific gravity, 1025; albumin, a trace; red blood-cells, leucocytes, squamous cells, small round epithelial cells. Apparently not as many pus-cells as before the operation. Albumin less in amount.

A few days later, the analysis showed a specific gravity of 1020, a trace of albumin, leucocytes, small and large round cells, quadrate and squamous cells.

December 16, 1910: The urine was of a specific gravity of 1017, acid, with no albumin or sugar, with very few leucocytes, and very few squamous cells.

December 27: The wound was solidly healed. There was no pain, though the patient had been up and about for several days.

On March 1, 1911, this patient was seen in my office (in response to a letter of inquiry), and was found at this time in excellent condition. There had been no pain. There was no tenderness. The kidney was palpable in the position in which it had been moored, and was not abnormally sensitive to touch. A specimen of urine obtained was clear, of 1020 gravity, and without a trace of albumin. Her general health was improved and might be described as fair, though still showing under-nutrition.

Since the body of this article was written, I have chanced to observe a case that may be of value by way of contrast, a case of hæmatogenous infection associated with a frank and fatal pyæmia.

CASE III.—M. O'N., female, aged twenty-four years, married, previously healthy, was admitted to the Boston City Hospital March 20, 1911, under the diagnosis of gall-stones. She had been well until four days previous, when she began to menstruate, and (contrary to her custom) began to vomit and to have severe pains in the abdomen just below the ribs. The pain and vomiting persisted; on the second day she began to be jaundiced.

On the fourth day she entered the hospital, very sick, showing a slight jaundice, marked right hypogastric and costo-vertebral tenderness, and a slight systolic murmur over the heart.

Investigation showed, apart from the mentioned findings, a red count of 2,064,000 with a white count of 47,000. At this time she showed slight mental "foginess." The urine showed albumin and few casts, nothing else.

Two days later the jaundice was less, the kidney tenderness was less, but the septic temperature and the septic anæmia persisted, and all means employed to check a hiccup which had developed had failed.

March 24, the local symptoms were less, the kidney evidently smaller and less obviously tender, but her condition in general was not improved.

March 27: Local signs less, but condition worse. Hiccough severe; vomiting occasional, but cannot be checked.

For 24 hours, more or less, she had been failing. At 4.30 P.M. she suddenly grew worse, and died within a few minutes in such fashion as suggested a terminal pulmonary embolism. No autopsy could be obtained.

This case showed a general sepsis. Dr. Libby (who examined her in consultation with me) found endocarditis, pericarditis, and a probable central pneumonia, beside the kidney lesion.

The case is brought up simply for contrast: it was a case of general sepsis with a kidney lesion apparently unilateral, but, owing to the generalized septic process, entirely beyond surgical reach.

The first case calls for little comment. It is typical for an early case of the type originally described, except for the complication of the floating kidney.

In the second case it may fairly be objected that no definite infarcts were found. Nevertheless, a septic infection of central origin seems clear from the clinical picture of sepsis presented, promptly and permanently relieved by relief of the extreme congestion found at operation. Engorgement alone will not account for the septic picture.

Whether such a case is a pure diffuse infection, or whether if left longer it would show localized infarctions, I do not know.

It is of interest that both cases were in "floating" kidneys, considerably displaced. In all the series so far recorded there is noted a preponderance of females over males, of multiparæ over nulliparæ, of infections of the right as against the left side.

No one seems to have noted, however, that all these differences coincide exactly with the conditions predisposing to *displacement* of the kidney.

My cases both showed definite displacement of the infected (right) kidney—how many previous cases showed the same condition is a question not to be answered from the

records. It is at least a striking coincidence, and perhaps more than that.

May there not be an explanation here of the point that has puzzled all the writers on this subject, namely, why the kidney (and the kidney only) should be the picked site of localization for an infection that must primarily have been a general blood infection?

If I am right in this surmise, then the operation done in these two instances represents an improvement in technic (in the earlier cases at least), for it must not only relieve tension and provide drainage, but must also relieve the abnormal position which is a cause of poor circulation and of abnormal intracapsular tension.

The recorded series of cases is too short to warrant any very positive conclusions, but it does seem undesirable to sacrifice a kidney because *part* of its substance is gone, if we can by any safe means save the rest of it.

In the later and severer cases the kidney may be past saving. In the earlier and simpler, and in the doubtful cases, it seems to me worth while to try and save what we can. Nephrectomy is easy and can be done later, if need be.

May it not be wise, in the doubtful cases at least, to try the measures of wide decapsulation, suspension, and drainage, that worked out so fortunately in the two instances here noted?

MALIGNANT PAPILLARY ADENOMA OF THE KIDNEY.*

BY J. BENTLEY SQUIER, M.D.,
OF NEW YORK.

THE large variety of renal tumors which have been histologically differentiated is evidenced in Küster's classification. Including those of the kidney proper, the capsule, and the adrenal, twenty-six distinct types are mentioned. Among these, malignant papillary adenoma is of sufficient rarity to justify the presentation of this patient and kidney specimen.

In a series of 529 cases of renal tumors, Albarran and Imbert found only thirteen malignant adenomata. Eisenstaedt, after a careful search of the literature, found one absolutely proven case, that of Sudeck in 1892, in addition to the one reported by him.

The history of the case which I desire to report is as follows:

The patient, a carpenter, forty-three years of age, appeared at my clinic March 9, 1911.

On December 1, 1910, while lifting a heavy weight, he was seized with severe pain in the left inguinal region, which radiated to the left lumbar region. A desire to urinate was present, and upon voiding, blood in large amount was noticed. For one week he was confined to his bed. During this time, the pain continued, he vomited frequently, and the hæmaturia slowly disappeared. At the end of the week, all symptoms had cleared up, and one week later he went back to work.

He was then without symptoms until February 1, 1911, when, on lifting a heavy weight, the symptoms returned. During this attack, several finger-like clots of blood were voided. The passing of the clots caused severe tenesmus. The duration of the attack was six days.

* Read before the American Association of Genito-Urinary Surgeons, June 2, 1911.

When he appeared at the clinic, he had had no symptoms for four weeks, with the exception of an occasional sensation of weakness in the left lumbar region. His past history was negative, except for some irregular gastro-intestinal symptoms extending over a period of years, such as flatulence, sense of weight in epigastrium, and constipation. He had always been a hard working man, and did not remember ever having had serious illness. His mother had died of some form of cancer.

Physical Examination.—Medium frame, fairly well nourished, skin and mucous membrane good color, no adenopathy, chest normal, abdomen negative, kidneys not palpable. Rectal examination negative. Cystoscopy showed a normal bladder, prostate, and ureteral orifices. Ureteral catheterization collected normal urine from either kidney. Radiographic examination of kidneys did not aid diagnosis.

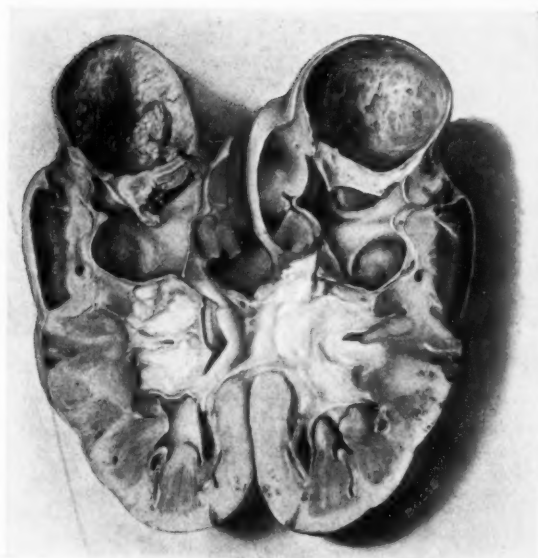
In view of these negative findings, the patient was sent home to be observed by his family physician. On March 18, a recurrence of the hæmaturia took place lasting two days. He was admitted to the hospital on March 21, and upon the following day, left nephrectomy was performed through a vertical lumbar incision. There were many adhesions about the upper lobe of the kidney. The wound was closed about a rubber tissue drain.

Convalescence from the operation was uneventful; the drain was removed at the end of 48 hours; the wound healed by first intention. He was out of bed at the end of a week, and left the hospital on April 3.

Pathological Examination.—Specimen: Kidney examined after fixation in formalin and opened by a longitudinal median section. At one pole there is a globular cystic cavity whose wall, in the hardened state, has a yellowish color, being thin over that portion of its extent which projects beyond the kidney parenchyma. The exposed portion of the wall of the cavity measures between 2 to 3 mm. in thickness, and the cavity is filled with yellowish granular material. Where the cyst is imbedded in the kidney substance, a soft, friable, narrow layer of grayish tissue separates it from the pelvis which is dilated at that point (Figs. 1 and 2).

Section A: Sections, including that portion of the wall of the cyst which is imbedded in the parenchyma of the kidney, present the following picture: The cyst wall is made up of a zone of connective tissue with numerous areas of lime deposit. The kidney parenchyma is bounded by this layer, except at one point where there is a tumor tissue just outside of the cyst wall, between the kidney tissue and the fibrous

FIG. 1.



Malignant papillary adenoma of kidney.

FIG. 3.



Low power microphotograph of Section A, showing normal kidney and cyst wall with tumor tissue between.

FIG. 4.



Low power microphotograph of tumor tissue.

FIG. 5.



High power microphotograph of tumor tissue.

membrane just described. Attached to and partly separated from the inner aspect of the cyst wall is some necrotic material, including slits that doubtless were filled with cholesterin crystals. These crystals must have been imbedded in a degenerate cyst content, the nature of which cannot be definitely ascertained, but which probably represents broken down tumor tissue, in view of the fact that the shadows of cells can be distinguished. At a point corresponding to those areas of the gross specimen which presented the yellowish material previously mentioned, there is a layer of papillary adenoma in which the proliferative phenomena, multiplication of cells, and formation of solid nests are sufficiently well marked to warrant a diagnosis of malignant adenoma. For the

FIG. 2.

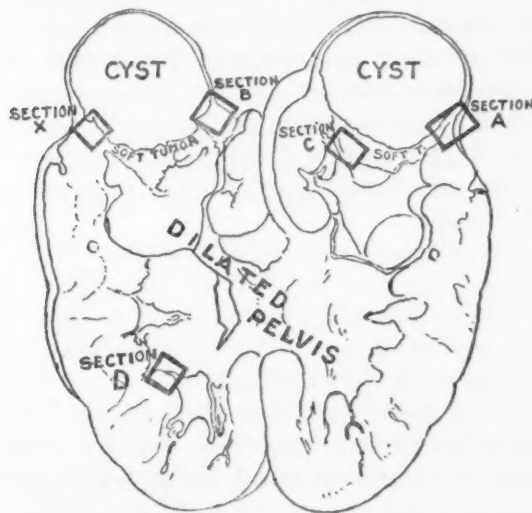


Diagram showing locations of sections examined microscopically.

most part in this area there are slender, tuft-like outgrowths, covered with cuboidal and polygenal cells, communicating and fusing with solid masses in a manner characteristic of those adenomata that show a transition into a medullary type. In the area outside of the cyst and next to the kidney parenchyma there is a similar papillary tumor. The kidney tissue itself at this point shows marked atrophy, foci of round-celled infiltration, and degeneration of glomeruli (Figs. 3, 4 and 5).

Section B: Another section taken from the border of the cyst shows only a minute area of papillary tumor in the fibrous wall itself.

Section C: Sections made from the soft tissue just outside of the cyst wall show papillary adenoma undergoing marked degeneration. In this specimen where the papillary outgrowths are not so crowded the cells take on a high cylindrical form.

Section D: A piece of the wall of the pelvis of the kidney shows moderate thickening, due in part to a submucous inflammatory process and œdema.

Diagnosis.—Cystic tumor of the kidney, in the wall and outside of which is a papillary malignant adenoma. Owing to the dense fibrous nature of the cyst wall, with its calcareous inclusions, it must be assumed that the cyst is the older lesion; that it may originally have harbored a benign papillary adenoma which has since become malignant (malignant adenoma), and is invading both the fibrous tissue wall of the cyst and the parenchyma of the kidney.

Since leaving the hospital and returning to work, there has been a continuance of the gastro-intestinal symptoms.

Considering the possibility of metastasis in the stomach, a series of X-ray plates were made of the stomach on May 25. The findings were as follows:

Stomach is of the cowhorn type. Size: Normal.

Position: Slightly prolapsed, the greater curvature being on a level with the umbilicus, the pylorus $1\frac{1}{2}$ in. above and to the right of the umbilicus.

Peristalsis: Of the 4-wave type, moderately active and equal in greater and lesser curvatures, not interfered with by adhesions or new growth of the wall of the stomach.

The first portion of the duodenum, which usually appears as a triangular cap, has more the appearance of a brass button on a steer's horn when the stomach is of this type. This triangular cap or button is separated from the pyloric end of the stomach by a space $\frac{3}{16}$ of an inch, which indicates the normal pyloric sphincter.

Diagnosis.—From a study of these plates, one is justified in stating that the stomach is of the size, shape, and position as described in the findings, moderately active, motor phenomena of the 4-wave type, with no evidence of new growth of or pressing on the stomach.

Conclusion.—The malignant papillary adenoma was probably primary in the kidney and localized.

POLYP OF URINARY BLADDER IN A THIRTEEN MONTHS OLD CHILD.

WITH A REVIEW OF THE LITERATURE.

BY IRVIN S. KOLL, M.D.,
OF CHICAGO.

THE rarity of vesical tumors in young children makes the subject one of the most interesting in genito-urinary surgery. Not only is the interest from a surgical and clinical aspect, but also from the pathological picture, which is quite varied. Mandelbaum¹ conveniently classifies neoplasms of the bladder according to their origin: (1) epithelial tissue group: papilloma, adenoma, carcinoma, cysts, polyps; (2) connective-tissue group: fibroma, myxoma, sarcoma; (3) muscle tissue group: myoma.

This division is more or less used with some variation in many of the pathological reports reviewed, covering the past twenty-eight years of literature.

In 300 cases of bladder tumor which came to operation, Von Frisch² reports only three in children—nine, eleven and thirteen years of age. All three of them were papillomata. Davis's³ youngest patient was sixteen years of age, in thirty-seven cases operated. Rumpel⁴ reports a case of myxofibroma in a male, three years of age, successfully operated, with no recurrence at the end of six months. Steffen⁵ has compiled thirty-one cases, covering a period of twenty-two years. Most of the specimens were obtained at autopsy. All of the thirty-one cases died. Twenty-one were sarcomata, one papilloma, nine myxoma. Fifteen of the cases were in males, sixteen in females. The average age was five years; the youngest eleven months (sarcoma), the oldest twelve.

Hüsler⁶ cites fourteen cases collected by Steinmetz, from the European clinics, from 1893 to 1905. The average age was five and one-half years; the youngest one and one-half,

the oldest twelve. Ten of the fourteen occurred in males. Seven of the tumors were benign, seven malignant. Of the benign he gives the following mixed classification: 2 fibroma-œdematosum, 1 fibrorhabdomyoma, 1 myxorhabdomyoma, 1 myxoma, 1 papilloma. The seven malignant tumors were all sarcomata.

Half of the number submitted to operation. Of these only three recovered; one had a recurrence one and a half years later, with fatal issue.

Clinically, the manifestations are more or less similar in all the cases. Two of Hüsler's cases, briefly cited, are as follows:

Male, one and one-half years; normal delivery. When four months of age mother on one occasion noticed a few drops of blood at end of urination. There followed a gradually increasing painful and difficult urination. An œdema and swelling of the external genitals developed; then suddenly complete retention, with a rapidly succeeding uræmia, and death. At autopsy an œdematous tumor mass, the size of a small apple, was found apparently springing from the urethral mucosa.

The second case was a male, seven years of age, whose mother, six months prior to his entrance to hospital, noted that it took the child a long time to urinate, and that the urine came away in dribbles. Later the patient began complaining of a sticking pain and burning when passing his water. This increased, when suddenly there was complete retention. Only once a small amount of blood was seen. A soft rubber catheter introduced into the urethra encountered a slight resistance at the entrance to the bladder. Per rectum under anæsthesia, a movable mass was palpated in the fundus of the bladder, which was about the size of a plum. Operation was refused. Obstipation developed; sudden perforation of bladder into rectum. Exitus. Autopsy showed a tumor of the size described obstructing the right ureteral orifice and extending up into the fundus. Microscopic diagnosis was myxoma-papillare.

The writer's case was sent to him by Dr. Lester E. Frankenthal, who had seen the patient in consultation with Dr. H. H. Frothingham. The mother gave the following history: From birth, which was instrumental, the patient, now thirteen months old, has been in perfect health up to one week before entrance into Michael Reese Hospital. At this time the mother noted that the child had not urinated for some hours and was crying, evidently from pain. On looking at his abdomen, she thought it appeared distended, and then sent for Dr. Frothingham, who

FIG. 1.



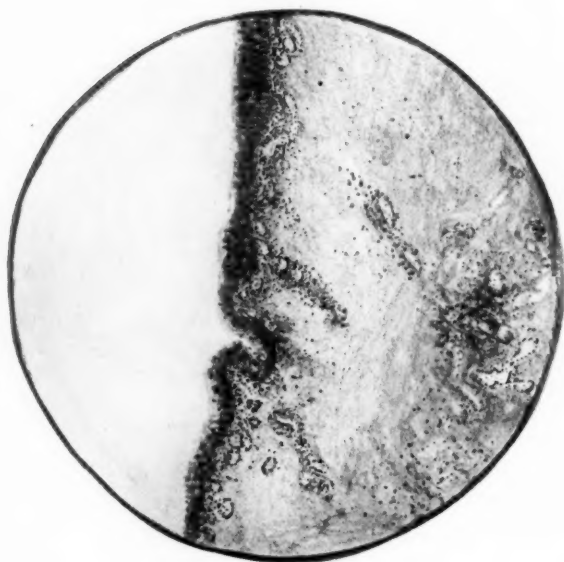
Polyp attached to internal urethral orifice.

FIG. 2.



Outline of tumor. Section through pedicle. $\times 20$.

FIG. 3.



Structure of tumor. $\times 103$.

catheterized the patient and obtained a large quantity of turbid urine. For a short time the child urinated small quantities, then again there was complete retention.

When I first saw the patient he was in great pain, the fundus of his bladder reaching as high as his umbilicus. There was a marked phimosis, with a very long prepuce. Catheterization yielded 360 c.c. of slightly turbid urine which, upon examination, showed a great many leucocytes, no blood and, culturally, a pure strain of colon bacillus. Upon passing the catheter a distinct obstruction was encountered at the internal urethral orifice, which, however, promptly allowed the catheter to pass into the bladder.

Cystoscopic examination showed only a slightly inflamed mucosa, no calculi and no neoplasm was seen. At no time was there any hæmaturia.

Circumcision and permanent catheter for forty-eight hours did not relieve the retention. At intervals it was noted that the patient would urinate a few drops, then suddenly stop, and he would use a great deal of abdominal muscular force in the vain attempt to empty his bladder. Rectal examination was negative. Careful exploration with a sound again encountered the resistance at the internal urethral orifice. The diagnosis of obstruction due either to some congenital malformation or a new growth was made, and consent for an operation was obtained.

The typical suprapubic incision was made. When stripping back the peritoneum a marked hypertrophy of the musculature of the bladder was very evident, and the bladder itself was about three times its normal size. As soon as the bladder was incised and the walls retracted, a tumor mass was seen, about the size of a small hazelnut, lying just below the internal urethral orifice (Fig. 1). This was smooth, had two lobules, and was attached by a pedicle, about 2 cm. in length, which sprang from the mucosa lining the posterior wall of the urethral orifice. This relation explains how it was that the patient could at times pass a small amount of urine, then have a sudden stoppage—a typical ball-valve action. The connection to the urethral mucosa also explains why the tumor could not be seen with the small cystoscope, but probably would have been visible had it been in an adult.

The growth was grasped in a pair of forceps and twisted

off its pedicle, this procedure being considered preferable to having any ligature material lying in such an undesirable position. There was no bleeding from the stump. The bladder was closed completely with two rows of running plain catgut. A very small fistula developed on the fifth day, which very promptly closed, the wound healing by primary union. Spontaneous urination took place immediately following the operation, and the convalescence has been uninterrupted.

The pathological report is that of a simple polyp, covered by a layer of stratified squamous epithelium, continuous with that of the mucosa of the urethral orifice and bladder. Magnification twenty times shows the outline of the tumor, with section through the pedicle (Fig. 2). Magnification 103 shows the structure of the tumor under a higher power (Fig. 3).

REFERENCES.

- ¹F. S. Mandelbaum: Pathology of New Growths of Bladder, Surg., Gynec. and Obst., 1907, v, p. 315.
- ²V. Frisch: Bericht über 300 Operierte Blasentumoren, Wien klin. Woch., 1907, xx, p. 1205.
- ³Lincoln Davis: Primary Tumors of the Urinary Bladder, ANNALS OF SURGERY, Phila., 1906, xliii, p. 556.
- ⁴Rumpel: Über kindliche Blasentumoren, Deut. med. Woch., xxxiv, p. 1855.
- ⁵A. Steffen: Die malignen Geschwülste im Kindesalter, pub. 1905 by F. Enke, Stuttgart.
- ⁶G. Hüsler: Beiträge zur Lehre von den Harnblasengeschwülsten im Kindesalter, Jahrbuch f. Kinderheilkunde, 1905, lxii, p. 133.
- ⁷Guizy: Trois Cas de Tumeurs de la Vessie sans Hematurie, Ann. d. Mal. des Org. Gen.-Urin., 1907, xxv, p. 103.

**INTRAPERITONEAL OPERATION FOR EXTENSIVE
CARCINOMA OF THE BLADDER WITH NEW
METHOD OF TREATING THE DIVIDED URETER.**

BY PAUL M. PILCHER, M.D.,

OF BROOKLYN, NEW YORK.

THE following case came to our private hospital for treatment through the courtesy of Dr. John W. Poole, of Brooklyn:

CASE REPORT.—A woman, sixty-two years of age. Family and previous history negative. Her first intimation that anything was wrong was the appearance of a small amount of blood in the urine ten months before consulting us. This means that the bladder involvement had already reached some advancement at that time. Blood continued to be passed in the urine at intervals, with some periods when no trace of blood could be found. Later the amount of blood in the urine has greatly increased, although it is absent at times. There was only occasional increased frequency of urination. Lost considerable weight, but still an obese, thick-set woman. No symptoms referable to either kidney.

Cystoscopy.—Right ureter opening (ostium) normal. Right kidney function normal, as shown by the phenolsulphonephthalein test (12 minutes).

Left ureter opening swollen and distorted, and seen to be involved in a carcinomatous growth. The opening itself was crater like, being surrounded by a raised up cauliflower growth.

The left ureter opening, the left wall of the bladder, a portion of the trigone, some of the anterior wall, and an area extending around the urethral orifice were seen to be involved in the malignant growth.

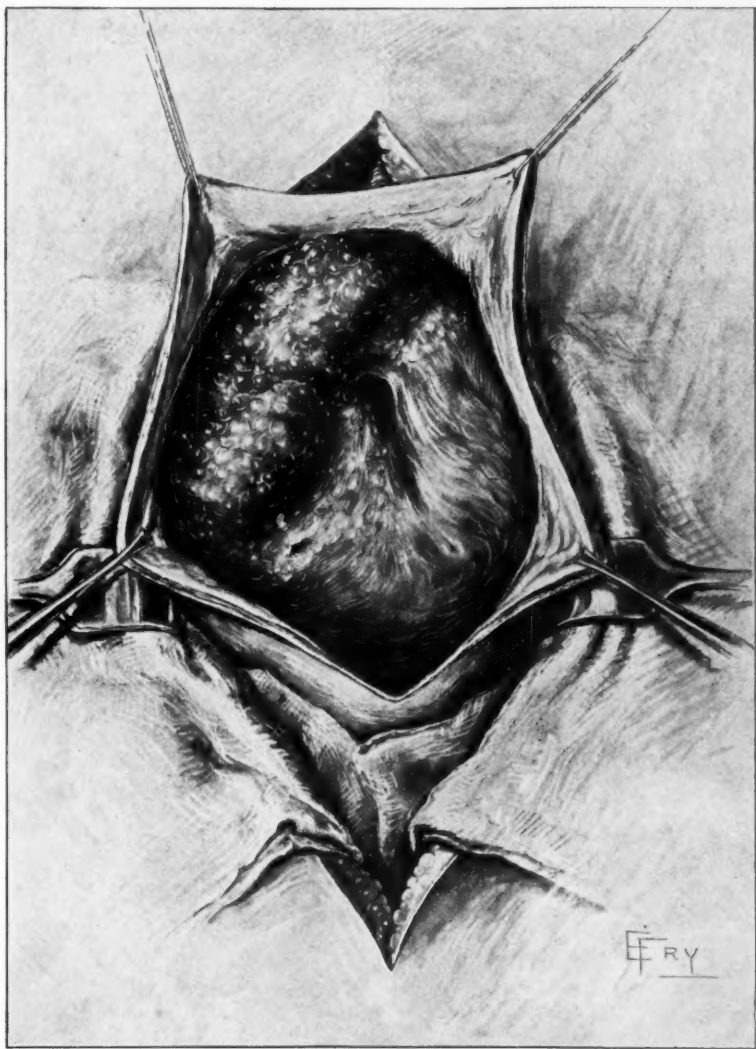
A vaginal examination revealed a distinct, hard mass occupying the vesicovaginal septum, about two inches in length by one inch in width. It did not involve the vaginal mucous membrane. Every one will recognize the extent of the lesion which was to be dealt with.

Operation, April 21, 1911, by the writer, assisted by Dr.

Lewis S. Pilcher and Dr. James T. Pilcher. Incision from umbilicus to symphysis; peritoneal cavity opened; Trendelenburg posture; pelvis emptied of intestines and protected by gauze pads; bladder widely opened in the median line and growth well exposed. Using scissors, the bladder wall which was involved was detached from the healthy bladder wall down to the urethral opening and the left ureter opening. At the point of exit of the left ureter it was found that the growth was superficial, having extended along the mucous membrane without involving the deeper structures. It had not apparently extended into the lumen of the ureter. The mucous membrane, the submucous structures, and a portion of the muscular tissue surrounding and underlying the left ureter opening including all of the tissues involved in the malignant growth, were freed and dissected up, the ureter itself not being cut across. By using traction on the flap which had been separated it was found that the ureter was loosened from its sheath within the bladder wall and could easily be pulled down through the bladder wall. In this particular case over two inches of normal ureter were drawn through the bladder wall into the interior of the bladder. The question of what should be done with the ureter was next to be solved. It occurred to me that a great deal of time would be saved and danger avoided, if, instead of transplanting the ureter, the ureter should be cut across *in situ*, about an inch from its ostium, and simply be allowed to slip back into place. This was done, the lumen cut across and the end split up about one-quarter of an inch to avoid narrowing the outlet by cicatricial contraction.

My theory was this: Having removed the lower inch of ureter and a portion of the interior of the bladder there would remain an interval of from one-half to three-quarters of an inch between the cut end of the ureter and the interior of the bladder. The bladder musculature having been injured, and the bladder being kept empty for three or four days, would offer very little resistance to the efflux of urine from the ureter along the avenue of least resistance, the course of the ureter through the bladder wall being changed from an oblique one to a much shorter and more direct one. Experience with ureterovaginal, uretero-abdominal, and other ureteral fistulas has taught that if we have an unobstructed ureter, then a shell of other tissue forming a sinus and then a surface lined with mucous membrane, and no other outlet for the

FIG. 1.



An extensive growth is shown after opening the abdomen, packing off the pelvic viscera with gauze and freely opening the bladder by a vertical incision, including the anterior and superior surfaces of the bladder. It will be noticed that the growth involves the left side of the bladder, more superficially the ostium of the left ureter opening and the first portion of the urethra. This growth also involves the vaginal septum, but not the mucous membrane of the vagina.

FIG. 2.

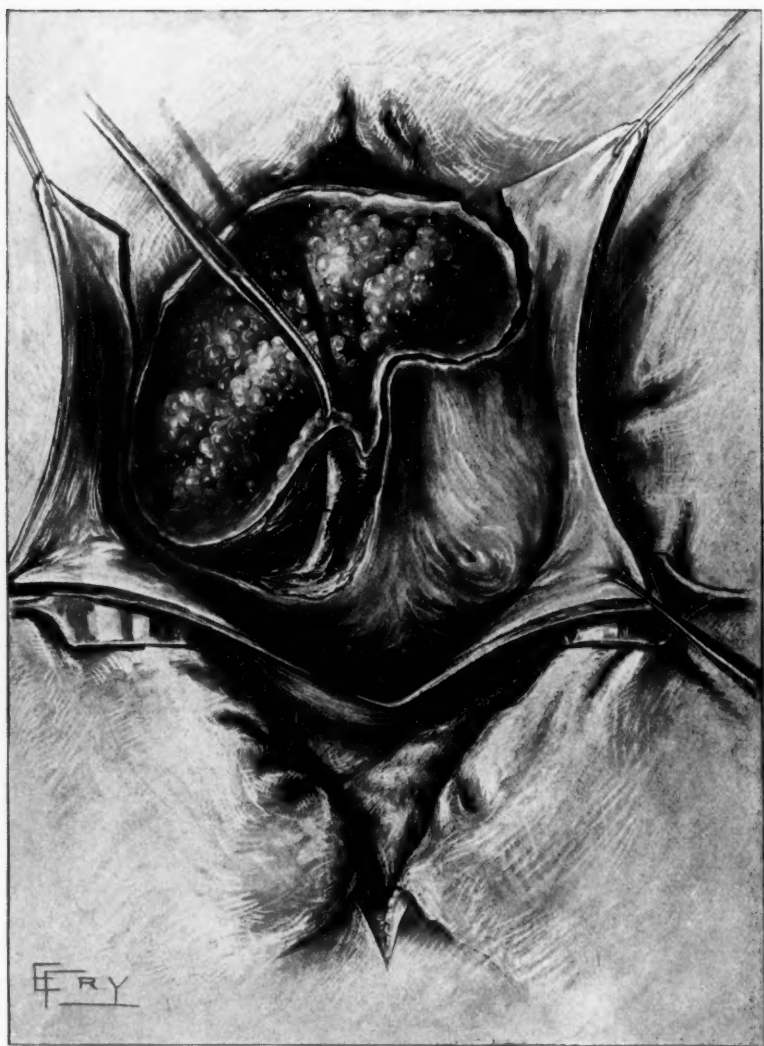


Fig. 2 shows the area of the bladder which was removed at operation, including the entire growth, most of the left lateral portion and inferior portion of the bladder, the left ureter opening and a portion of the urethra. The area above the left ureter opening has been dissected up. Here the growth was superficial. In raising up this portion of the bladder wall, including the opening of the left ureter, it was noticed that the ureter itself was pulled through the bladder wall, and about two inches of the lower end of the ureter extended into the wound, free from carcinoma. Instead of transplanting the ureter, it was cut across as indicated in the drawing by a transverse and vertical line about an inch from its ostium, and the severed end of the ureter was allowed to slip back into place, after the end had been slit up about one-quarter of an inch to avoid narrowing of the outlet. There remained then an interval of one-half inch between the cut end of the ureter and the interior of the bladder, and a ureterovesical fistula was established.

urine from the ureter, the establishment of a fistula along this avenue is absolutely certain. I made use of this knowledge in the present case and intentionally formed an *ureterovesical fistula*.

After all the cancerous tissue had been removed, including much of the surrounding tissues beneath the symphysis, a portion of the anterior vaginal vault and all but a section of the urethral orifice, the bladder was reconstructed. The vesicovaginal opening was not closed and a rubber drainage tube was led through it from the bladder to the vagina. The anterior and lateral walls were brought together first by a running Connell suture of catgut and reinforced by a running Lembert suture of catgut. The intraperitoneal portion of the bladder was closed by a separate suture and reinforced *à la Mayo*. The peritoneal cavity was closed without drainage. The suprapubic portion of the wound was closed with the exception of a cigarette drain behind the symphysis. A second drain of rubber tubing was brought out beneath the symphysis and to the left of the labium majus. The bladder had in this way been entirely closed excepting at its base in the region of the trigone and to the left of the urethral orifice, which could not be entirely covered in but which was satisfactorily drained by the tube in the vagina. The left kidney discharged its urine via the left ureter, then through the ureterovesical fistula into the bladder.

The patient made an uneventful operative recovery. There was no leakage of urine excepting through the vesicovaginal opening. The drainage tube was removed from beneath the symphysis on the third day; from the suprapubic opening on the fourth day; the vesicovaginal tube on the fifth day. At no time was there any symptom referable to the left kidney. She passed urine *per urethram* on the tenth day. The leakage *per vaginam* ceased about the sixteenth day. She left the hospital on the thirty-first day, completely recovered.

I appreciate that the first criticism which would be offered would be that the bladder musculature would contract and stenosis around the vesical orifice of the fistula take place. Our experience, however, in cases of prostatectomy in which a comparatively large section of the prostatic urethra has been entirely removed without the development of any obstructive

stricture at the vesical outlet led me to hope that a similar process would protect the ureterovesical opening.

The experience gained in this case has led me to theorize as to the possibilities of attacking the lower end of the ureter through the bladder, not simply to remove calculi, as has been so frequently done, but in the treatment of strictures of congenital origin or any disease involving the lower end of the ureter which does not involve the peritoneum. It is essential to my mind that the peritoneum should be intact, for, even if a relatively large portion of the ureter were removed or stripped out from its peritoneal covering, the tunnel thus left would suffice to convey the urine to the bladder and form an ureterovesical fistula. Knowing how difficult it is, oftentimes, to cure an ureteral or a vesical fistula of accidental origin, I think we would be justified in taking the chance of the fistula remaining patent. I believe that the chances of this are just as good, and not attended by so many chances of failure, as in ureteral transplantation in the hands of the majority of surgeons. In the only case of transplanted ureter which has come under my observation, post-operatively, I found that no urine was entering the bladder from the corresponding kidney.

SPLENECTOMY FOR RUPTURE OF SPLEEN.*

WITH REPORT OF FOUR CASES.

BY JOHN C. McCOY, M.D.,

OF PATERSON, N. J.

SPLENECTOMY is indicated in certain pathological conditions affecting the spleen causing an enlargement of the organ, interfering with the health of the individual, or for the reason that an enlarged spleen is a source of danger to the patient, owing to its susceptibility to laceration from trauma or even rupture without external violence.

The hypertrophy may be due to various pathological changes in the spleen substance, or the enlargement may be the result of systemic or infectious processes. Among the former causes may be mentioned: tumor of the spleen due to cysts, parasitic and non-parasitic; malignant growths, usually sarcoma, either primary or secondary; benign growths; abscess of the spleen, and contusion. No well-authenticated case of primary carcinoma is recorded.

Of the systemic and infectious conditions giving rise to enlargement of the spleen are malarial and syphilitic spleen; amyloid spleen; hypertrophy of the spleen with cirrhosis of the liver, and tubercular spleen. It has been generally accepted, that tuberculosis of the spleen never occurs as an independent condition, but is usually secondary to a general tuberculous process, and in itself is of very little clinical importance. So far as a differential diagnosis between a tuberculous enlargement of the spleen and a hypertrophied spleen from other pathological conditions is concerned, I believe the differentiation would be impossible in the absence of tubercular symptoms in other organs. That it is possible to have a tubercular condition present in the spleen with no apparent tubercular process elsewhere in the body, is illustrated in one of my cases.

* Read before the New Jersey State Medical Society, June 15, 1911.

The spleen may be classed as one of the blood-producing organs, and probably takes an active part in disposing of the broken-down blood-cells. How far it may exert an inhibiting effect, acting in the capacity of a mechanical filter, upon organisms entering the circulation, we cannot say. It has been claimed that patients from whom the spleen has been removed are more susceptible to infection. In no cases reported, however, has the removal of the spleen been followed by conditions which could be attributed to its absence. As to the effect upon the economy the extirpation of the spleen has, it is hard to say. It would seem that in those cases in which there were no marked pathological changes in the organ, there is no detrimental effect in the individual as the result of the removal of the spleen.

In two of my cases of extirpation following traumatism of the spleen, which have been carefully followed since operation, in the one case for a period of 16 months and in the other for 7 months, the general condition of the patients seems to have remained as good as it was prior to the loss of the organ. Both patients have steadily gained in weight, are able to indulge in athletic sports, and show no evidence of fatigue, even under severe exertion. Blood examination demonstrated a practically normal condition, 16 and 17 months respectively after splenectomy. It has been noticed in both cases, that though there has been a steady gain in flesh, and an excellent capillary circulation, giving an almost florid complexion, there is a faint but evident underlying bronze hue to the skin over the entire body, resembling the early stage of hæmatogenous jaundice of pernicious anæmia. Could this be explained on the basis of the absence of the spleen, which prior to its removal had disposed of the broken-down cell elements of the blood? We know from animal experimentation, and observations upon the human subject, following the removal of the spleen, that its presence is not essential to life; neither does its extirpation seem to interfere with the physical well being of the individual.

As to how far other parts of the economy, such as the

bone-marrow or lymphoid tissues, take on increased activity after removal of the spleen, is not definitely known. There has been observed in some instances, after splenectomy, a general enlargement of the lymphatics and enlargement of the thyroid. We know that during fetal life, the spleen forms red blood-cells, this function being probably stopped at birth. It has been stated that the bone-marrow assumes increased activity upon removal of the spleen and compensates for its absence.

In cases of rupture of the spleen, in which extirpation has been done, the observations and subsequent blood examinations would seem to show that the changes in the blood elements were more marked and permanent than would be expected from the simple loss of blood. From our own investigations, it would seem to be only a question of time, when the blood constituents again reach a normal state.

Certain changes in the blood appear to be constant following splenectomy: (1) an increase in the number of white blood-cells; (2) a diminution in the number of red-cells; (3) an increase in the number of eosinophiles; (4) a disproportionate and more persistent diminution in the hæmoglobin.

Cases of rupture of the spleen are not common, 160 cases having been reported up to 1908. A large percentage of ruptures have occurred in pathological spleens, mostly in spleens enlarged from malarial or infectious conditions.

During the past three years, I have seen four cases of rupture of the spleen, two of which could be attributed to direct violence. The spleen was removed in each instance with one fatal result, that of the gunshot case.

The first case was one of gunshot wound of spleen. Case II was one of contusion of an apparently normal spleen, with subsequent rupture. Case III was one of contusion of a malarial spleen, with subsequent rupture. Case IV was a spontaneous rupture of a tubercular spleen with absolutely no history of trauma, appearing in a patient while in bed asleep.

CASE I.—Mrs. M., age thirty-eight. Seen in consultation with Dr. George Fisher. During a period of mental depression, placed a 32-calibre revolver against the left hypochondriac region and discharged the ball into the abdomen.

Physical Examination.—Large robust woman, married. Heart and lungs normal. Very fat abdominal wall. Pelvic examination negative. Perforating wound just below the free border of the ribs on left side three inches to the left of the ensiform cartilage, skin surrounding wound showing powder marks. Perforating wound just below border of ribs in anterior axillary line of left side, evidently point of exit of bullet. Abdominal wall rigid on right side and sensitive to pressure over left hypochondriac region; most marked just between the ribs. Temperature 98°; pulse 90, and there was nothing apparently alarming about her condition.

During the next 48 hours, the abdominal rigidity became more marked, distention more pronounced. There was evidence of general peritonitis. At end of 48 hours, temperature 102°, pulse 120; vomiting. Urinalysis negative.

Operation.—Median incision, free blood in peritoneal cavity, general peritonitis, rupture of the spleen, bullet having passed directly through the organ. Splenectomy. Death in 36 hours.

PATHOLOGICAL REPORT BY DR. F. R. SANDT.

The organ was but slightly increased in size, it was firm to the touch, the capsule was normal, the surface smooth, and the edges sharp. One portion of the organ, about one-fifth of it, was completely detached by the passage of a bullet and the rupturing effects of the resulting hemorrhage. Microscopical sections gave no pathological changes except in area close to injury, where there was an extensive hemorrhagic infiltration of the splenic tissue.

As stated, it has been claimed that tuberculous enlargement of the spleen *per se* does not occur. Dr. W. J. Mayo in 1909 reported a case of splenic tuberculosis, in which, other than the enlarged spleen, the physical examinations were negative. I wish to add the following case of splenic tuberculosis terminating in spontaneous rupture of the organ, occurring in a woman, who up to the time of the rupture of the spleen had been in her usual health, and showed no evidence of tubercular infection after the removal of the spleen.

CASE II.—I am indebted to Dr. Rush Neer, with whom I saw the case in consultation, who had the case under observation for two days before admission to the hospital, for the following statement: "Patient went to bed Feb. 7, 1909, feeling perfectly well. About 4 or 5 A.M., Feb. 8, awoke suddenly with severe abdominal pain. When seen by me, the abdomen was distended, dulness on both sides, especially painful over left curvature of the colon. Purgatives administered and hot stupes applied to the abdomen.

"Feb. 9, several evacuations of the bowels, less abdominal distention, less pain. Vomited. Temperature 99.6°; pulse, 104.

"Feb. 10, temperature 99°; pulse 120. Severe abdominal pain, more distention."

Seen in consultation with Dr. Neer, Feb. 10, 2 P.M. Entered hospital, 4 P.M.

Mrs. F., German, age sixty-seven, married 38 years, two children. States that five or six months ago was conscious of slight pain in the left side, particularly upon exertion; also noticed a more frequent desire to urinate. Other than the frequent urination and slight pain in left hypochondriac region, previous personal and family history negative. During the past two months, the frequency of urination has been more troublesome. Always constipated. Ceased menstruating at 47.

Examination.—Well nourished, very anæmic, skin bronze-like hue, conjunctiva jaundiced, facial expression anxious. Tongue thickly coated and dry. Heart and lungs normal. No enlargement of the lymphatics.

Abdomen: Prominence of the left side, extending from free border of ribs to pelvis. Palpation showed a more or less irregular mass, rather boggy in this area. Dulness extended from near the centre of the abdomen to the posterior axillary line. Pain on pressure was most pronounced over the region of the left kidney. Pelvis negative. Temperature 99°; pulse 102; respiration 36.

During the next 24 hours there was an almost constant desire to urinate, patient passing 22 ounces of urine in this time. Urine showed albumin, pus, and casts.

Feb. 10 to 12: Temperature ranged from 99° to 101°, less pain in abdomen, but prominence more pronounced. Amount of urine for 24 hours did not exceed 26 ounces. Urine continued to show blood and pus.

Feb. 14: Cystoscopic examination showed evidence of chronic cystitis. Ureteral catheter introduced into the left ureter and left *in situ* six hours. Diagnosis of pus kidney made, and operation advised, but refused by family.

Feb. 14 and 18: Patient's condition seemed to improve, began to take light food, and bowels moved daily. Less abdominal pain, and diminution of abdominal distention. The mass diminished in size, extending to within two inches of the umbilicus and to within three inches of iliac crest. Extremely tender over entire left side. Rigidity of right rectus. There continued to be frequent desire to urinate, which seemed to be relieved when patient was placed in semireclining position. The low 24 hours' urine persisted, varying from 18 to 24 ounces; each examination showing blood and pus.

On the morning of Feb. 19, patient had a severe chill, lasting 15 minutes, followed by a rise of temperature to 102.4° , pulse 128. Tumor in left side more prominent and distention more pronounced. Pain excruciating.

Operation at 1 P.M., Feb. 19. Longitudinal incision in lumbar region from ribs to ilium. When kidney was reached, it was found about one-third larger than normal, surface apparently normal. Large intraperitoneal hæmatoma presented. Peritoneum opened, when there was evacuated three or four quarts of chocolate-colored blood and clots. When cavity was cleansed, there presented an enlarged spleen having several lacerations of its capsule, upon its upper surface. An incision extending inward from the upper extremity of the original cut was made, giving freer access to the spleen. Spleen was then turned over. Splenic vessels clamped and ligated and spleen removed. Patient's condition was serious; 1200 c.c. normal salt solution given intravenously.

Two days after the operation, the temperature was 99° ; pulse 86. From this time convalescence was uneventful, the patient leaving the hospital on the thirty-fifth day after operation. The maximum temperature during convalescence was 99.4° and maximum pulse 98. The maximum amount of urine passed in any 24 hours during this period was 41 ounces.

The patient remained in fairly good health for 11 months after leaving the hospital, when she died, from what the attending physician designated as pneumonia. It would seem fair to pre-

FIG. 1.



Contusion of apparently normal spleen; subsequent rupture (Case II).

FIG. 2.



Contusion of malarial spleen; intracapsular hemorrhage; rupture (Case III).

FIG. 3.



Tuberculous spleen; spontaneous rupture (Case IV).



sume that the cause of death was probably pulmonary tuberculosis. It is also probable that the lesion in the left kidney was tubercular. The peritoneum and intestines exposed appeared normal.

The pathological report of the spleen, signed by Dr. F. R. Sandt, is as follows:

Specimen of a Spleen.—Case of Mrs. F. The organ measured 25 cm. in length and 16.5 cm. in width. Weight 450 Gms. The specimen was uniformly enlarged and the surface smooth. To the touch there was a doughy feeling as if a cystic condition existed. The capsule was thin and stripped easily; three ruptures of the capsule through which the splenic pulp protruded were noted. A longitudinal section of the organ showed a firm periphery, but the central portion was partially destroyed and converted into a number of small irregular communicating cavities, the walls of which were composed of the fibrous tissue of the spleen or the trabecula. Microscopic sections from the periphery gave the picture of an acute splenitis. The capsule was thin, no trabecula and Malpighian corpuscles were visible. The cells were closely packed together, and four types of cellular elements were found. First, the cells of the splenic pulp, large granular elements similar to the large mononuclear cells of the blood; second, small lymphocytes; third, narrow sickle-shaped cells; and fourth, polymorphonuclear leucocytes. A fatty degeneration of some of these cellular elements was noted. Sections from the deeper portions show a number of imperfectly formed and degenerating tubercles. Numerous giant-cells were noted. These tubercular processes were surrounded by densely packed lymphocytes. Tubercle bacilli were demonstrated in these areas, within the giant-cells, and from the broken-down material from the interior of the cavities.

Diagnosis.—Acute hyperplastic splenitis secondary to a chronic tuberculous splenitis.

The third case demonstrates the effect of trauma upon an apparently normal spleen, the injury resulting in a contusion of the organ, causing a laceration of the splenic pulp, and producing an intracapsular hemorrhage. The tension upon the capsule becoming more and more intense, finally terminated in a rupture of the organ four days after the reception of the injury.

CASE III.—*Intracapsular contusion of spleen, followed by rupture of organ.* A male, fifteen years old, an American, was admitted to my service with the following history: Three days before admittance to hospital, while coasting down a hill of moderate grade, in attempting to avoid a bob sleigh, left side of

abdomen was struck by fore runner of sleigh. He was able to arise, walked one-half mile to his home, complaining only of a slight pain in left side of chest and abdomen. This pain, however, gradually became more intense, becoming localized to left superior quadrant of abdomen, and was constant. On account of the increasing severity of the pain, twelve hours after the accident a physician was summoned, who treated the case palliatively for three days, during which period the patient was slightly prostrated and presented only one symptom, that of pain. Urination was normal. There were no gastro-intestinal disturbances.

Physical examination showed a young, fairly well-developed man, markedly anæmic, prostrated, restless, respirations thoracic. Head, neck and chest were normal. The abdomen was uniformly distended; abdominal respiration was absent. There was rigidity of both recti, most marked on left side. Tenderness was present over entire epigastrium. Dulness existed on left side, extending anteriorly to within one inch of anterior axillary line and extending upward to seventh intercostal space. On the right side of abdomen, dulness reached anterior axillary line. Dulness did not shift.

Temperature was 102° ; pulse 98, regular fair volume; respirations were 22. About 18 hours after entering the hospital, pulse suddenly rose to 132, became irregular, small, and tension was below normal. Respirations rose to 28, and temperature fell to 101° . Patient became restless, jumped out of bed, abdomen became suddenly distended, and patient went into a condition of partial collapse.

On account of history of injury, of syndrome of symptoms presented, with localization of pain in left superior quadrant of abdomen, a diagnosis of ruptured spleen was made. In view of the fact that the patient had passed only 11 ounces of urine in the past 18 hours, the possibility of rupture of the kidney was also considered, although there were no pathological urinary findings.

Four hours after condition of partial collapse had come on, patient was operated upon. A median incision 10 inches long was made over middle abdomen. Abdominal cavity contained large amount of dark blood. The upper angle of incision was prolonged laterally under free border of ribs. Spleen was found lacerated and about twice normal size. In order to control hemorrhage, it was found necessary to extirpate the organ.

Patient, beyond suffering from shock subsequent to operation, made an uneventful recovery and was discharged four weeks after operation. June 9, 1911, examination showed him to be in perfect physical condition; has gained 24 pounds in weight since the accident. During past spring has entered several canoeing contests in long-distance races.

PATHOLOGICAL AND BLOOD REPORT BY DR. F. R. SANDT.

Specimen of Spleen.—J. W. The specimen measured 16.25 cm. in length, 11.25 cm. in width, and weighed 392 Gm. The organ was slightly enlarged, surface smooth, firm to the touch, capsule normal, edges sharp. A ragged tear extended over the anterior surface of the spleen, involving the capsule and splenic tissue to about the centre of the organ.

Microscopical Examination.—No pathological changes were found in the tissue except in the area about the traumatism, where there was an extensive hemorrhagic infiltration.

Diagnosis.—Rupture of the spleen and traumatic hemorrhagic splenitis.

Blood Examinations.—On admission to the hospital on Feb. 4, 1910, an examination of the blood showed the following condition: red cells 3,408,000; leucocytes 19,000; hæmoglobin 70 per cent.

Differential count: small lymphocytes, 10 per cent.; large lymphocytes, 15 per cent.; polymorphonuclear forms, 74 per cent.; eosinophiles, 1 per cent.

After operation on Feb. 5, 1910, the count was as follows: red cells, 2,472,000; leucocytes, 15,600; hæmoglobin, 30 per cent. Differential count had not materially changed: small lymphocytes, 16 per cent.; large lymphocytes, 12 per cent.; polymorphonuclear cells, 69 per cent.

On Feb. 8, 1910, three days after operation, the maximum changes were found. At this time the red cells reached their lowest point, 2,300,000; the leucocytes attained their highest point, 30,800, and the hæmoglobin was present only to the extent of 20 per cent. Differential count: small lymphocytes, 16 per cent.; large lymphocytes, 9 per cent.; polymorphonuclear forms, 75 per cent.

A gradual improvement in the blood state now ensued; the red count gradually increased, the leucocyte count fell to the neighborhood of 18,000, and the quantity of hæmoglobin slowly increased. One month after operation a count gave 4,320,000 red cells, 19,500 leucocytes, and 85 per cent. of hæmoglobin. The differential count gave 26 per cent. of small lymphocytes; 18 per cent. of large lymphocytes; 54 per cent. of polymorphonuclears, and 1 per cent. of eosinophiles.

Three months after operation, on April 15, 1910, the count was as follows: red cells, 4,600,000; leucocytes, 17,200; hæmoglobin, 85 per cent. Of the leucocytes, 20 per cent. were of the small lymphocytic variety, 6 per cent. were of the large forms, 3 per cent. were of the transitional varieties, 62 per cent. were polymorphonuclear, and 9 per cent. were eosinophilic. This count gave us the maximum number of eosinophiles.

On March 9, 1911, 13 months after operation, a count gave 4,720,000 red cells, 16,200 leucocytes, and 90 per cent. of hæmoglobin; 18 per cent. of the leucocytes were of the small lymphatic variety; 10 per cent., large lymphocytes; 3 per cent., transitional; 67 per cent., polymorphonuclears, and 2 per cent. eosinophiles.

On June 9, 1911, the results were as follows: red cells 5,560,000, leucocytes 15,800, and hæmoglobin more than 100 per cent. The differential count gave 16 per cent. small lymphocytes; 11 per cent. large lymphocytes; 9 per cent. transitional forms; 63 per cent. polymorphonuclears, and 1 per cent. eosinophiles.

Conclusions.—No permanent blood changes were found in this case. Sixteen months after operation a normal condition with a slight increase in the number of leucocytes was found. The leucocytes in this case seem to persist at a slightly higher level than normal. The hæmoglobin was restored more quickly, and the normal color index was found sooner than in a case to be subsequently reported.

The eosinophilia, which has been noted by other observers as following splenectomy, was only moderate in this case and developed two months after operation. No structural changes in the red cells were ever noted in any of the specimens examined.

The fourth case demonstrated the effect of traumatism upon a pathological spleen, which was probably enlarged as the result of a chronic malarial infection, the traumatism causing a contusion and laceration of the splenic pulp and the accompanying intracapsular hemorrhage. The rupture occurred 48 hours after the injury. It is of interest to note the recurrence of a malarial infection following the injury to the spleen, and the fact that, although a short course of malarial treatment was instituted at this time, there has been no recurrence of the malarial trouble since the attack occurring 10 days after the splenectomy, nor have the malarial organisms been found at any time during the past four months.

CASE IV.—Male, Holland, age twenty years, painter, while working on a roof fell from a height of 40 feet, striking left side on ground. Patient was unable to arise, was semiconscious; complained of pain in left shoulder. He was immediately taken to hospital, November 9, 1910.

Past History.—Patient has had frequent attacks of malaria, last attack being six months prior to accident. Was treated for chills and fever for two months.

Physical Examination.—With exception of slight abrasion about one inch long on forehead, head was normal. Neck and chest normal. The left shoulder drops forward, downward, inward. There is a diffuse swelling over anterior end of clavicle, over which tenderness and crepitus can be elicited. Abdomen shows only slight rigidity of left rectus, most marked lower portion where patient complains of pain on pressure. Spleen not palpable. Liver normal. No masses, no fluid made out. Pulse 110; respiration 24; temperature 99.6°. General condition was fair. Patient vomited a few times; vomitus, food and bile. About 18 hours after admission, patient went into a condition of collapse. The pulse became small, rapid, 136; tension low; respiration 24; temperature 100.8°. Abdomen slightly distended, absolutely rigid, tenderness diffuse over entirety. On account of marked rigidity and tenderness definite signs were not obtainable. Rectal examination showed mass in pelvis, which was soft and fluctuated. A Leiter coil was applied over abdomen and Murphy drip given.

About 48 hours after admission patient went into a condition of collapse; pulse became very rapid and irregular, respirations were shallow, thoracic, had a peritonitic facies and appeared markedly anæmic. Abdomen appeared board-like.

On account of history of injury, of anæmia present, together with set of symptoms presented, a diagnosis of rupture of the spleen was made. Urination being normal and there being no pathological urinary findings, rupture of kidney was not considered.

Four hours after the symptom complex had become aggravated or 52 hours after reception of injury, patient was operated upon. Incision about five inches long made along border of left rectus in upper two-thirds of abdominal wall. A large amount of chocolate-colored blood was found in peritoneal cavity. Incision was prolonged from upper angle of wound along free border of ribs and downward from lower, for distance of three inches.

Spleen was found lacerated and about four times normal size. In order to remove organ, it was found necessary to incise

costal cartilage of tenth rib near chondrocostal junction. After ligation of vessels, abdomen was flushed out with warm normal saline. A stab wound was made in flank and cigarette drain inserted. After recovery from shock of operation, patient made rapid recovery, although 15 days after operation he had a chill, rise of temperature to 103° , pulse 128, followed by profuse sweating.

Two days later, temperature rose to 103.4° , pulse 130, preceded by chill and followed by sweating. This yielded to quinine. Plasmodia found in blood. Forty-two days after admission, patient was discharged, cured.

From this time convalescence was uninterrupted. Plasmodia were found in blood 15 days after admission. Have not been found since that time, although there have been frequent examinations of the blood, the last examination made June 10, 1911.

PATHOLOGICAL REPORT AND BLOOD FINDINGS BY DR. F. R. SANDT.

Specimen of Spleen.—S. D. The specimen measured 20 cm. in length, 1.25 cm. in width, and weighed 375 Gms. The organ was somewhat enlarged, edges rounded, surface wrinkled, capsule thickened, and adherent. A laceration was noted extending over the middle of the spleen, dividing the organ for about two-thirds of its extent. The microscopical sections show a thickened capsule, prominent trabecula, and prominent Malpighian corpuscles. The splenic pulp was not distinct, and in sections stained with iodine very small mahogany-brown areas were found. The central artery of the corpuscle was normal, but the finer vessels leading from it through the corpuscle show waxy changes. Sections from the area near the traumatism show hemorrhagic infiltration.

Diagnosis.—Rupture of the spleen; beginning amyloid degeneration of the spleen, which is probably a secondary result of a malarial condition.

Blood Examinations.—A blood count made after admission to the hospital on November 10, 1910, resulted as follows: red cells, 4,000,000; leucocytes, 17,000; hæmoglobin, 70 per cent.; the differential count being normal, showing 20 per cent. of small lymphocytes; 4 per cent. of the large mononuclears; 5 per cent. of transition forms; 68 per cent. polymorphonuclears and 2 per cent. eosinophiles.

On the morning of November 12, 1910, before operation, the red cells were found to number 3,136,000, leucocytes, 37,000, and the hæmoglobin had dropped to 40 per cent.

On the evening of November 12, 1910, after operation, there was a further reduction in the red cells to 2,496,000; the leucocytes numbered 25,000, and the hæmoglobin was reduced to 35 per cent., which was the lowest point reached in this case. The number of red cells were also lower at this time than at any subsequent time. On this date there was a marked change in the differential count, the average number of cells,

as based on the two counts, giving 4 per cent. lymphocytes; 3 per cent. large mononuclears; 0.5 per cent. transition forms; 92.0 per cent. polymorphonuclears, and 0.5 per cent. of eosinophiles.

On November 13, 1910, the red cells were found to number 3,000,000; the leucocytes, 37,600; and 40 per cent. of hæmoglobin; 87 per cent. of the leucocytes being polymorphonuclear.

No material changes were found in the blood examinations, with the exception of the increase of the hæmoglobin to 50 per cent. until November 23, 1910, when the red cells dropped to 2,736,000 and the highest leucocytosis of the series, 39,600, was recorded; at this time the polymorphonuclear cells were present to the extent of 88 per cent. Nothing unusual was noted on this date, when the stained preparations were examined for the differential count, and the rise in temperature, when associated with a leucocytosis of 38,600, was attributed to a possible infection.

The periodicity and the degrees of temperature elevation on November 24, with no local evidence of suppuration, led to another examination of stained preparations, and at this time tertian malarial parasites were easily detected. They were so numerous that from four to five organisms could be found in a single microscopic field.

On November 28, 1910, after a daily administration of quinine, the red cells were found to number 3,040,000; the leucocytes, 9,200; and the hæmoglobin remained stationary at 50 per cent. At the time of this examination, the differential count showed 39 per cent. of small lymphocytes; 23 per cent. large lymphocytes; 2 per cent. transition forms; 32 per cent. polymorphonuclears and 4 per cent. eosinophiles.

From this date on until January 16, 1911, there was not much change in the blood findings, except that the red cells were gradually increased in number and the percentage of the various forms of leucocytes again became normal. The hæmoglobin did not vary, but remained constant at 50 per cent.

On January 16, 1911, the red cells had increased to 5,024,000; the leucocytes numbered 17,000; and the hæmoglobin registered 65 per cent. The differential count at this time resulted as follows: small lymphocytes, 14 per cent.; large mononuclear forms, 15 per cent.; transition forms, 5 per cent.; polymorphonuclear forms, 55 per cent.; eosinophiles, 8 per cent.; myelocytes, eosinophilic in character, 3 per cent.

The increase in the number of eosinophilic cells reached their maximum on March 6, 1911, when 14 per cent. of the leucocytes were of this form. On this date, the red cells were practically normal at 4,800,000; the leucocytes, 5,880; hæmoglobin, 70 per cent.; polymorphonuclear forms, 45 per cent.; and 17 per cent. small lymphocytes.

Subsequent counts in the case gave no additional points of interest. The final count in the case was made on June 6, 1911. The results were as follows: red cells, 5,456,000; leucocytes, 11,600; hæmoglobin, 90 per cent.; the differential count showed 15 per cent. small lymphocytes; 6 per cent. large mononuclears; 7 per cent. transition forms; 70 per cent. polymorphonuclears, and 2 per cent. eosinophiles.

The points of interest in the blood findings in this case were: 1. The absence of any marked reduction in the red cells following the operation, other than that caused by the hemorrhage resulting from the traumatism and the effects of the dilution of the blood by the saline transfusion, and the absence of any of the abnormal forms of red cells; poikilocytes, nucleated red cells, and abnormally large or small cells were not noted at any time.

2. The slow regeneration of the hæmoglobin; a low color index was noted at all examinations up to the date of the last one, when it was found to be 0.99. For three months following operation, the hæmoglobin remained constant at 50 per cent., even though the number of red cells had gradually increased to practically normal.

3. The occurrence of a moderately severe leucocytosis immediately following the hemorrhage for which operation was performed, which reached its maximum ten days after operation (39,600) and which then rapidly declined to practically normal within another week.

4. The gradual development of a moderate eosinophilia, which reached its maximum about four months after operation and which then fell to the normal limit.

5. The appearance in the blood of an active malarial process ten days after operation, at a season when there was no possibility of a fresh infection by the *Anopheles*. With the history of an active malaria during the preceding summer, can this be explained on the basis that the parasites were dormant in the spleen and were liberated into the general circulation at the time of the traumatism and hemorrhage?

6. The absolute restoration of the blood in quality, as evidenced by the normal count obtained seven months after the removal of the spleen.

In neither of the two cases which have been carefully watched since operation has there been changes in lymph-glands, enlargement of the thyroid, or pains in the long bones.

The diagnosis of contusion of the spleen causing an enlargement of the organ from intra-capsular hemorrhage can

only be made upon the previous history of trauma. There is no symptom characteristic of the condition, nor would it be possible to differentiate from a tumor of the spleen due to other pathological changes, which might have been present prior to the injury. A patient showing a tumor and evidencing pain: the splenic area following trauma in this region, particularly if accompanied by abdominal rigidity, should be carefully observed for subsequent rupture of the spleen. The diagnosis of rupture of the spleen is difficult, whether the rupture occurred at the time of injury, or the traumatism was the result of the contusion and subsequent rupture.

When rupture has occurred, the clinical picture does not differ materially from intra-abdominal hemorrhage due to rupture of other organs. It is not always possible to differentiate between rupture of the spleen and rupture of the kidney. The pressure resulting from the accumulation of blood from a ruptured spleen may cause urinary symptoms closely resembling those found in rupture of the kidney. In rupture of the kidney, we have found in several cases that on careful rectal examination there could be distinctly felt an elevation of the posterior parietal peritoneum of the left side, due to hæmatoma from the ruptured kidney, which symptom is absent in splenic hemorrhage.

I am indebted to Dr. F. R. Sandt for the careful blood observations made in these cases.

PROLAPSED SPLEEN WITH ACUTE TORSION; SPLENOPEXY.

WITH TABLE OF UNREPORTED CASES TO DATE.

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CASE REPORT.—Mrs. W., housewife, seen in consultation with Drs. E. N. Libby and J. P. McCue. Three months ago was in hospital with an attack similar to present, but much less severe. Several slight attacks since. Patient would not go out, fearing pain would come on while away from home. No history of malaria. Four days ago patient was taken with a sharp pain in left hypochondrium, cramp-like in character, and so severe that it caused her to vomit. Pain and vomiting have persisted, with only short intervals of relief. Between attacks there is no desire to vomit. The pain is said to be much worse when lying on her right side, better when on back, and better still when holding on to lower abdomen, but touching her upper abdomen is impossible on account of "sore place" just to left and above umbilicus. She states that the pain is never in front, but always in the left side, beneath ribs. The pain is in no way referable to food, and is not relieved by vomiting. There has been no blood in the vomitus or urine. The bowels have moved well at all times, and there has been no passage of urine in relation to the attacks.

The patient is a well-developed and nourished woman. Heart, lungs, and general examination negative, except for the abdominal findings. Just below rib in left hypochondrium there is considerable tenderness, but practically no spasm of the rectus can be made out. On deep palpation a smooth, round mass may be felt, which is movable downward as far as the umbilicus and displaces easily on pressure outward toward the loin. It is acutely tender, and any degree of pressure causes the patient intense pain. On account of this tenderness it is not possible to palpate the tumor bimanually in the loin. It is apparently solid in consistency.

The white count is 8400; temperature has never been above 100°; and the urine is negative.

A diagnosis of prolapsed spleen or kidney was made, with a feeling from the ease with which it displaced toward the loin that it was a loose and twisted kidney.

Operation was advised and accepted. A lumbar incision was made and the kidney pulled into the wound. It was found only slightly movable and it was evident that it was not the tumor felt in the abdomen, as with the hand in the kidney pouch it was possible to still feel the smooth tumor through the unopened peritoneum. The kidney capsule was split and a few stitches inserted to hold it in place. The loin wound was closed and the patient turned upon her back. The abdomen was then opened through the left rectus muscle, just below the costal margin, and a large slaty-red mass dotted with flakes of fibrin brought up into the wound by the examining hand. It was evident at once that the mass was an enormously enlarged spleen, and on closer examination it was found that the broad pedicle of parietal peritoneum was so twisted on itself as to almost completely shut off its venous return. Upon the surface of the spleen were numerous fine flakes of fibrin and at one pole a small accessory spleen. On examination it was evident that the spleen was still quite closely attached to the abdominal wall at its inner pole, and that it had practically rotated upon this attachment.

The liver, kidneys, stomach, and transverse colon seemed in normal position, and were not prolapsed.

The spleen was untwisted, returned to its normal position, and fixed there by four large stitches of chromic catgut, passed through the loose peritoneum close to the spleen, which formed the pedicle, and then with the intestines well packed off, into the tissue covering the parietes, endeavoring in each case to include periosteum in the grasp of the needle. These stitches were tied, the accessory spleen removed for pathological examination, and the abdomen sutured in layers.

On coming out of ether, the patient no longer complained of pain, nor has she at any time since—now four and a half months after operation. Her recovery was uneventful, the wounds healing by first intention. She was not allowed to leave the reclining position for approximately one month. Since that time she has been about her affairs with no return of symptoms in the slightest degree.

The pathological report of sections taken from the accessory spleen was normal spleen tissue, except for enormous congestion.

An examination of the abdomen at this time, four and a half months after operation, finds the spleen just palpable below the costal border, and not movable to any extent from side to side.

Etiology.—Prolapse of the spleen occurs as a part of general enteroptosis, or may be caused by congenital anomalies, such as abnormal length of the ligaments or by acquired lengthening, the result either of trauma or increased weight of the spleen, due to malaria, leukæmia, or pseudoleukæmia. As a proof of the congenital origin of the relaxation of the splenic ligaments, Osler cites its occurrence in different members of the same family.

It is an interesting fact that, although prolapse of the spleen occurs commonly with general enteroptosis, in a majority of the cases operated upon and reported, nothing is said of ptosis of the other organs, and in this case, as is stated in the history, the abdominal organs were in apparently normal position.

Diagnosis.—The diagnosis of prolapsed spleen is made from the shape and position of the organ, and may be confirmed by palpation of its edges and by the most confirmatory sign of all, the feeling of the splenic notches. In certain instances it is said that the vessels entering the spleen may be palpated. The diagnosis is also aided by ruling out kidney prolapse after ascertaining that both kidneys are in normal position. In most cases where the diagnosis has been made, the splenic notches have been felt.

Symptoms.—In all cases of torsion there is great pain, due to dragging on the pedicle from the additional weight of the congested spleen, stretching of the capsule from the same cause, and the occurrence of an acutely tender mass, as a rule movable in all directions unless surrounded by adhesions. Temperature, pulse, vomiting, and shock are dependent upon the degree of torsion.

Treatment.—The indications are to remove the twist and to

restore the organ to as near normal conditions as possible with a reasonable prospect of its remaining so, and if this is not possible, to remove it. In the list of cases reported since June, 1908, only one is spoken of in which splenopexy was done, and in this case gauze packing was also used. In another the spleen was simply untwisted and no attempt made to restore it to place; and in another, gauze packs and a belt were used. In the remaining cases splenectomy was practised. There were no fatal results in any of the methods of treatment.

In nearly all of the treatises on surgery, splenectomy is recommended and splenopexy condemned because of the proneness, it is stated, of the prolapse to recur in cases with a marked degree of descent. That this is true seems more than likely, yet in view of the lessened danger, the somewhat hazy idea which we still have about the spleen, and the success of the simple procedure in these three cases and in other cases reported in the earlier literature, it seems that there must be a place for splenopexy, either by suture, packing, or pouch. In a certain group of cases similar, I believe, to the case here described, in which all of the supporting structures of the spleen are not yet gone and the spleen has not prolapsed into the lower abdomen, also in those cases in which time is of the greatest value on account of previous shock or exhaustion from pulling on the splenic pedicle or the suffering which goes with it, untwisting the pedicle and suturing into as near normal position as possible, with packing or not, will be the most rational treatment, and the place most suitable for splenopexy.

In those cases where the spleen is in the pelvis, or low down in the abdomen, with a long and corded pedicle making splenectomy rapid and simple, it will be the operation of choice, with only slight danger and an absolute guarantee of non-recurrence.

In this case to have removed the spleen would have been a much different problem than the simple removal of a spleen with a long pedicle, and would have added a degree of risk that would have been unjustifiable.

In an article on splenectomy, George Ben Johnson reports

the occurrence, prior to 1890, of splenectomy for torsion five times, with four deaths; between 1890 and 1900, eleven times, with four deaths; and between 1900 and 1908, eleven times, without a death.

Below is submitted the cases of prolapsed spleen with torsion reported in the literature since that date (June, 1908); six treated by splenectomy without a death; one by simple untwisting resulting in recovery; one by untwisting and packing; two, including the author's, by splenopexy, resulting in recovery.

C. R. Blackburn and R. G. Craig. *Australian Med. Gaz.*, Dec., 1907, vol. xxvi, p. 615. Replaced from pelvis and held by pads. Good recovery.

Asa B. Davis. *Bull. of the Lying-In Hosp. of N. Y. City*, vol. v, No. 1, p. 24, June, 1908. Splenectomy while pregnant. Recovery and normal delivery.

J. E. Gemmell. *Jour. of Obstetrics and Gyn. of British Empire*, London, Oct., 1908. Untwisted and not removed on account of patient's condition. Seen a year later without symptoms.

J. E. Gemmell. *Jour. of Obstetrics and Gyn. of British Empire*, London, Oct., 1908. Splenectomy. Recovery.

Adam Reuterskiöld and Artur Vestberg. *Upsala Läkareförenings Förhandlingar*, June 20, xiii, No. 5, p. 355. Splenectomy. Recovery.

P. Paterson. *London Lancet*, No. 20, 1909. Splenectomy. Recovery.

I. MacDonald and W. A. Mackay. *London Lancet*, Sept. 25, 1909. Splenectomy. Recovery.

W. J. Mayo. *Jour. A. M. A.*, vol. liv, No. 1, p. 14. Suture and packing. Recovery.

CYSTS OF THE OMENTUM.*

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CYSTS of the omentum are uncommon; they give two main varieties of interest: (1) practical, referring to their diagnosis and treatment; (2) academic, referring to their origin.

1. From the practical stand-point we may remember that at rare intervals these cysts are found, that their symptoms in non-malignant cases are mainly mechanical, due to pressure or traction on surrounding structures, and that operations have in the main been successful and free from serious difficulty, about 86 per cent. of the reported cases having ended in recovery.

2. Origin: The origin in most of the cases has been apparently similar to that of mesenteric cysts. In a discussion of mesenteric cysts held before this society several years ago, the members assented to their classification into: (1) embryonic cysts; (2) cystic malignant disease; (3) echinococcus cysts.

Most of the cysts of the omentum may be included in the same classification, for it is evident that embryonic rests may occur between the peritoneal layers of the omentum as they do between those of the mesentery, although such occurrence is less common.

Cystic malignant disease and echinococcus cysts are occasionally found in the omentum, but their consideration does not properly come within the limits of this paper.

A number of very able articles have been written on omental cysts, among which we may particularly mention that by Hasbrouck.¹ By its aid and with a pains-taking search of the literature, Doctor Farr has prepared the following table.

* Read before the New York Surgical Society, March 22, 1911.

TABULATED DESCRIPTION OF OMENTAL CYSTS, EXCLUDING ECHINO-

Number	Age	Sex	Result	Symptoms	Cyst Wall					Lining			Contents					
					Thick	Thin	Unilocular	Multilocular	Closed sac	Fluid in folds of omentum	Endothelium	Epithelium	Fibrous	Clear or light colored fluid	Dark colored fluid	Coagulated blood	Milky	Pus
1	Adult	F.	D.	Not given.						+			+					
2	44	M.	D.	Anuria(?).		+ 1mm		+		+			+			+		
3	58	F.	C.	Like ovarian cyst.												+		
4	19	F.	C.	Pain at menses. Vomiting Shooting pain Ailing gen'ly.			+	?		+					+			
5	4	F.	C.			+	+						+	+	+			
6	48	M.	C.					+					+		+			
7	22	F.	C.	No pain. Sudden growth. Emaciation.		+	+				o		+	+				
8	2½	F.	C.						+							+		
9	Adult	F.	C.													+		
10	8	M.	C.	Dyspnœa Frequency.					+							+		
11	4	F.	C.	None		+	+							+				
12	7	F.	C.	Emaciation.		+	+		+		+			+				
13	1 yr	F.	C.	Pain		+			+				+		+			
14	8 mo.	M.	D.	Diarrhœa.				+										
15	21	M.	D.	General condition bad.	+											+		
16	1	F.	C.	Anorexia. Vomiting. Constipation.		+		+						+	Rose			
17	11	M.	C.	Vomiting. Pain. Anorexia. Emaciation.		+	+	?								+		
18	9½	F.	C.	Emaciation.		+		+								+		
19	2½	F.	C.	Indigestion.						+						+		
20	17	F.	C.	None but pressure.			+						+	+	+			
21	6	M.	C?	Pain.		+	+	?					+	+		+	+	
22	19	F.	C.	Pain. Tenderness.		+	+						+	+		+	+	
23	4	M.	C.	Emaciation.		+	+			+			+	+		+	+	
24	15	F.	C.			+		+			+		+	+		+		
25	51	F.	C.	Vomiting.		+							+	+		+		
26	51	M.	C.	Emaciation.		+							+	+		+		
27	4½	F.	C.	Gastro-enteritis.		+		+			+		+	+		+		
28	Child	M.	C.			+	+						+		+			
29	50	F.	C.			+		+			+		+	+		+		
30	4	M.	?	None.		+		+			+	+	+	+		+		
31	38	F.	D.			+		+			+	?		+		+		
32	50	M.	D.			+		+					+		+			
33	8	F.	C.	Pain. Vomiting.		+	+											+
34	26	F.	C.					+										
35	37	F.	C.	None. Pain.		+	+							+		+		
36	43	F.	C.	Vomiting. Dyspnœa.		+	+									+		
37	8	M.	C.	Vague pain. Dyspnœa.			+	?						+		+		
38	62	M.	C.	Dyspnœa. Emaciation Weakness.		+										+		

COCCUS CYSTS AND CYSTIC MALIGNANT DISEASES. BY CHAS. E. FARR, M.D.

Remarks	References
Autopsy specimen	Gairdner, W. T.: Trans. Path. Soc., London (1850-51), 1852, iii, 374.
Present 12 years. Autopsy specimen	Simon, E.: Bull. Soc. Anat. d. Paris, 1858, xxxiii, 30.
Many years	Doran, Alban: Trans. Obst. Soc., London, 1882, xxiii, 164.
Followed blow. Present 1 year	Gooding, J. C.: Lancet, London, 1887, i, 311.
Contained 4 pints	Wells, Spencer: Brit. Med. Jour., 1890, i, 1362.
No trauma. [Small cell sarcoma in wall.] Gradual development	Cazin, M.: Bull. Soc. Anat. d. Paris, 1893, lxxviii, 312.
Vascular inner wall. Contained 9 litres (partly necrotic material)	Erdheim, S.: Wien. klin. Rundschau, 1896, x, 131.
Large hemorrhagic	Marfin, A. B.: Press. Med., Paris, 1896, 133.
Albuminous fluid and cholesterin	Jessett, F. B.: Brit. Gynec. Journ., 1896-7, xii, 156.
Abdomen measured 44 in. Omental cyst diagnosed	Hearn, W. J.: ANNALS OF SURGERY, 1897, xxv, i, 703.
Indescent patches; 3½ pints	Braithwaite, J.: Lancet, London, 1898, ii, 1472.
Ten pints	Jacobi, A.: Trans. Assoc. Am. Phys., 1901, xvi, 232.
Blow on stomach 3 mos. before. Communicated to stomach	Marsh and Monsarratt: Brit. Med. Journ., London, 1901, i, 511.
Few red blood-cells	Catman, H. H.: Brit. Med. Journ., London, 1902, i, 1267.
Had a lining membrane. Attached to pancreas; 12 pints	Schramm, H.: Zentralb. f. Chir., 1903, xxx, 564.
32 pints	Boyd, S.: Clin. Journ., London, 1903, xxi, 306.
Bloody fluid	Young, W. McG.: Lancet, London, 1905, i, 157.
66 lbs.	Port, R. E.: ANNALS OF SURGERY, 1907, xlv, iii, 382.
Smooth lining membrane	Rodman, J. S.: ANNALS OF SURGERY, 1909, xlix, 427.
Unaltered blood; short duration; 2½ litres	Gifford, A. H.: Brit. Med. Journ., London, 1910, i, 1289.
"Lymphangiectasis"	Holleman: Zentralblatt f. Gynäk., 1908, xxxii, 297.
Bloody fluid	Seefisch, G.: Deutsche med. Wchenschr., Leipsig, 1909, xxxv, 1790.
Reddish jelly-like, partly calcified wall	Brandt: Zentralbl. f. Gynäk., 1894, v, 991.
By Czerny	Wagener: Nederl. Tijdschrift v. Geneesk., 1902, dl. 2.
Chocolate colored fluid. Clinic of Prof. Signori	Ris: Bruns Beiträge z. klin. Chir., x, 1893, 423.
Died later—autopsy	Schwarzenberger, B.: Bruns Beiträge z. klin. Chir., Tubing., 1894, xi, 713.
Noticed 2 years	Costa, Tomasso: Giornale internaz. del Scienze, Med., 1908, xxx, 357.
Some ciliated cells in lining. Autopsy case (pyæmia)	Himmelheber, K.: Archiv. f. Gynäk., 1909, lxxxvii, 67.
Autopsy specimen. Contents homogeneous	Wakefield, W. F. B.: Trans. Am. Gyn. Ass'n, 1907, xxxii, 447.
Suppurating dermoid; 3 plates of bone; pus	Karás, Henrietta: Virchow's Archiv., Bd. clxxxviii, 138.
Present 6 years; much ascites; 75 lbs.; fluid too thick to run	Henke: Verhandl. d. deutsche path. Gesell., Bd. ii.
No trauma; wall partly gangrenous	Waldy, J.: Lancet, 1889, vol. ii, 642.
Few blood-cells; no epi- or endothelium; lesser omentum	Ormsby: Med. Press and Circular, London, 1883, vol. xxv, 258.
No trauma; few blood-cells; lymphangioma	Edebohl: New York Journ. Obst. and Gyn., 1893, vol. iii, 614.
Cystic tumor	Schwartz, Rodolfo: Gazet. degli Ospedali, 1902, vol. xxiii, 764.
	Mathews, Paul: Brit. Med. Journ., 1905, vol. ii, 1642.
	Bidwell, Leonard J.: Brit. Med. Journ., 1905, vol. ii, 806.

It will be seen that most of the cases have been very incompletely reported, and that there have been few exhaustive descriptions of the cyst walls and the cyst fluid, hence it is impossible to reason with accuracy as to their origin. Apparently most and possibly all of these cysts were of embryonic origin. One must, however, notice how large a proportion of them gave evidences of blood in their contents—four of them contained coagulated blood, eighteen contained fluid so dark in color that they had apparently contained blood.

It is self-evident that an ordinary hæmatoma in the omentum should not be described as a cyst, but the results of hemorrhage into the omentum may well receive further study and it is believed that the case here reported may throw some light on the subject.

CASE HISTORY, No. 657 B.—G. R., aged twenty-six, a clerk and football player, admitted to Roosevelt Hospital, June 30, 1910. Discharged cured, July 8. Diagnosis, cyst of omentum.

Chief Complaint.—Lump in abdomen.

Present Illness.—Fifteen months ago while straining at stool, he felt something "snap" inside of him, and immediately began to feel very faint. He collapsed for several minutes, then recovered, then went to bed for ten days on account of weakness and pain in abdomen. He was nauseated and vomited once or twice. After lying in bed for ten days he felt well again. About four months ago he noticed a lump in the left side of the abdomen about half as large as a hen's egg. This lump apparently increases and diminishes in size; he thinks it is slowly getting larger. There has been constantly a dull aching pain in the tumor. No pulsation noted. Patient is habitually constipated. No fever. Appetite good. Has had acute attacks of cramp-like abdominal pain, about six attacks.

Past History.—Negative for infectious or contagious diseases except measles. Cardiorespiratory, negative. Gastro-intestinal, no stomach symptoms. Constipation. Abdomen too full, patient says. Urinary, negative. No gonorrhœa or lues. Alcohol in moderation.

Family History.—Negative to neoplasm or tuberculosis. Father died of stomach trouble.

FIG. 1.



Diagrammatic drawing of omental cyst with torsion of its pedicle.



Physical Examination.—Patient is a well-nourished young man, who does not look to be acutely ill. He shows no abnormality excepting in the lower part of the abdomen, where a tumor can be felt. It is $2\frac{1}{2}$ inches below the umbilicus and extends to the left from the median line. It seems about as large as an orange. It is firm, smooth, and apparently is not in anterior abdominal wall. Firm pressure over it causes pain. It is somewhat movable. No pulsation can be made out. No murmur is heard. Pressure seems to make no difference in size of tumor.

Operation.—Anæsthetic: gas and ether. Median incision. A cyst about as large as a grape fruit was found attached to the pelvic peritoneum; it had a pedicle above, which was composed of twisted omentum (see diagram). Its wall was thin. The tension of the fluid was moderate. A fold of peritoneum extended upward on to its anterior wall; when this was peeled down the cyst wall proper was found to be continuous with the omental peritoneum. The fluid was aspirated and found to be clear, thin, and watery. The cyst was then separated from its pelvic adhesions; its pedicle was ligated, and it was removed. On section the cyst wall was found to be composed of omental peritoneum and fibrous tissue into which this peritoneum was incorporated. Within this wall and pressed firmly against it there was a friable layer of coagulated fibrin. The patient made an uninterrupted recovery.

Pathological Report (Dr. Mortimer Warren).—The tumor was a cyst about the size of a large orange, and contained 8 to 10 oz. of clear fluid. It was covered with peritoneum. Beneath the peritoneum there was a rather thick layer which was bluish-green in color and very friable, breaking on being touched. The pedicle of the tumor was composed of mesentery twisted on itself many times.

Microscopical Examination.—Showed that the cyst wall was composed of fibrous tissue congested and infiltrated with round cells. It contained many blood-vessels and showed evidences of inflammation. The inner layer was coagulated fibrin and showed no definite cellular structure.

The cyst fluid was pale in color, sp. gr. 1008, albumin 1 per cent. (in bulk). There were a few blood-cells which apparently got into the fluid during the operation. Their examination showed a ratio of 216 red blood-cells to 36 white blood-cells.

A differential count showed: 4 per cent. polymorphonuclear cells; 60 per cent. lymphocytes; 36 per cent. endothelial cells. The fluid was apparently a transudate, not an exudate. There were no bacteria.

Probable Method of the Formation of the Cyst.—The facts that this cyst wall was little more than thickened peritoneum and that there was a layer of coagulated fibrin within it, lead one to wonder whether it could have been the result of an omental hæmatoma. Furthermore, the patient's history suggests such a formation. The attack came on suddenly while he was straining, he felt as though something had given way, he fainted and had to be in bed for several days, he had been subjected to frequent traumatisms, and hence was more apt than the average person to have omental hemorrhage. One who believes the cyst to have been the result of hemorrhage should explain the absence of hæmoglobin in its contents, the very light specific gravity of its fluid, and the apparent increase in the size of the cyst and in the pain.

Absence of Hæmoglobin in Fluid.—Adami and Bradley have explained the disappearance of the hæmoglobin.

Adami,² writing of hemorrhagic cysts, says: "Extensive hemorrhages into the substances of sundry organs may result not in the ultimate absorption of the exuded fluid but in cyst formation. The hemorrhage leads to the destruction of the tissue of the infiltrated area; eventually a capsule is found around the exuded blood, but while this is proceeding, through the combined agencies of leucocytes and autolysis the bodies of the corpuscles and the cell débris undergo removal, as does also the diffused and altered hæmoglobin, so that, after a few weeks, the cyst is found to contain a thin, blood-stained fluid, and eventually all the contents come to be a clear serous fluid. The last indication of the hemorrhagic origin of such a cyst is the presence of modified blood pigment in and around the fibrous capsule. The organs in which such hemorrhagic cysts are specially liable to be found are the brain, goitrous thyroid, scalp of the newborn and children (cephalhæmatoma), pinnæ of ears (football players and lunatics) (othæmatoma)."

Bradley³ has studied the same process in the thyroid gland, and has described seven thyroid cysts which showed

hemorrhagic origin and illustrated different stages of cyst development and absorption of hæmoglobin.

Brain cysts which were the sequelæ of hemorrhages and which contained clear fluid have been very often described, and they offer very good examples of the process under consideration.

Light Specific Gravity of the Fluid.—In speaking of the fluid, Dr. Warren said that it seemed like the fluid of œdema, not like the fluid ordinarily found within a cyst. Its specific gravity, 1008, was much less than that of blood, which is about 1060. Its content of albumin, 1 per cent. (by bulk), was also a small fraction of that in blood-serum, which is about 50 per cent. when estimated by the same method.

Can the light character of this fluid be explained by the twisted pedicle of the cyst? One may well believe that it can. Much has been written about torsion of the omentum. In 1900 Wiener⁴ tabulated seven cases; in 1905 Corner and Pinches⁵ 53 cases, and in 1910 Finsterer⁶ 72 cases, and records of a few other cases are to be found in the literature. Most of the cases (90 per cent.) have been associated with hernia.

The omentum has been twisted between two fixed points in 23 per cent. of the cases (Pretzsch⁷), its lower edge has been free in 77 per cent. This torsion is not so common as that of ovarian cyst pedicle and probably not so common as volvulus of the intestine. Either the existence of an omental hæmatoma or the adhesion of the omentum to the pelvic peritoneum would give conditions for omental torsion, and the existence of such a torsion in this patient is not to be wondered at. Torsion might easily cause œdema below the pedicle, and in a cyst with indefinite vascular walls this œdema might easily determine the character of the contained fluid; the watery elements being present in a larger proportion than in ordinary cysts.

The symptoms of torsion of the omentum have usually simulated those of strangulated hernia or appendicitis (sudden pain, nausea or vomiting, constipation, tenderness, rigidity,

etc.), and the attacks of pain and the discomfort which this patient had were probably in large measure due to the omental torsion.

Summary.—The cyst formation in this case is believed to have been due to the following elements:

1. Hemorrhage into the greater omentum, forming a hæmatoma.

2. Absorption of the hæmoglobin and degeneration of the blood-cells, as described by Adami and Bradley.

3. Torsion of the omentum, as in cases tabulated by Finsterer and others, with consequent œdema and transudation of the watery elements of the serum into the cyst cavity.

The attacks of pain were similar to those which have been described by patients with omental torsion.

The condition must be a rare one, but in these days of careful study of abdominal conditions, it surely has a practical as well as an academic bearing.

REFERENCES.

¹ Hasbrouck: *ANNALS OF SURGERY*, 1908, vol. xlviii, p. 207.

² Adami: *Principles of Pathology*, 1908, vol. i, p. 794.

³ Bradley: *Journal of Experimental Medicine*, vol. i, p. 401.

⁴ Wiener, Jos., Jr.: *ANNALS OF SURGERY*, vol. xxxii, p. 648.

⁵ Corner and Pinches: *American Journal of the Medical Sciences*, vol. xxx, p. 314.

⁶ Finsterer: *Brun's Beiträge zur klin. Chir.*, 1910, vol. lxviii, p. 52.

⁷ Pretzsch: *Brun's Beiträge zur klin. Chir.*, 1906, vol. xlvi, p. 118.

LINITIS PLASTICA (CIRRHOSIS OF STOMACH).

WITH A REPORT OF A CASE CURED BY GASTRO-JEJUNOSTOMY.

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THE term *linitis plastica* was used by Brinton to designate a special disease of the stomach, benign in nature, characterized, pathologically, by a diffuse or circumscribed increase in the connective tissue involving chiefly the submucosa, and to a lesser degree the other layers, giving rise to a marked thickening of the stomach walls with a corresponding diminution in its lumen; clinically, by its insidious onset, its slow progressive gastric symptoms, its cachexia, and fatal termination.

Nomenclature.—In searching the literature the following synonyms for the disease are found: (1) chronic interstitial gastritis; (2) sclerosis of stomach (Sneller); (3) fibroid induration (Handfield Jones); (4) fibroid disease of pylorus (Haberson); (5) hypertrophy with induration (Cruveilhier); (6) submucous hypertrophy; (7) submucous sclerosis with chronic gastritis and callous retroperitonitis (Hanot and Gombault); (8) hypertrophic stenosing gastritis (Boas); (9) hypertrophic stenosis of pylorus (Lebert); (10) fusocellular sarcoma (Lyonnet); (11) plastic linitis (Brinton); (12) cirrhosis of the stomach; (13) endogastritis obliterans (Leersum); (14) Magenschrunpfung; (15) Zuckergussmagen; (16) hypertrophy of the stomach (Fox); hypertrophic sclerosis of the submucosa (Bouvert); (17) atrophy of stomach (Fenwick); (18) Cirrhotische Verkleinerung des Magens (Nothnagel); (19) cancer conjunctif sous-muqueux (Bard et Garret); (20) neurofibromatose carcinomatose (Curtis); (21) gastro-intestinal sclerostenosis (Krompecher).

History.—The condition of scirrhus of the stomach had been recognized two to three centuries before Brinton's description. These early descriptions were scattered and incomplete, Lieutaud (1779), Voegtel (1804), Pohl (1804). In the second quarter of the nineteenth century the observations became more frequent. In France, Andral (1835), Cruveilhier (1835), Monnert (1841), Salse (1844), Bérault (1847), Lebert (1845), Boyer (1848), Broca (1850) described the condition. Andral's, Cruveilhier's, and Broca's descriptions are clear-cut, and definitely state that the lesion is benign. Lebert recorded a case in which the stomach, colon, and rectum were involved.

In Germany Gluge (1850) described a case of complete involvement of the stomach, Brant (1851), one in which the lesion was found in the stomach and cæcum. By these authors the lesion was held to be benign. In Austria Rokitsansky (1859) stoutly maintained that the submucous hypertrophies were nothing else than fibroid cancers.

To Brinton (1854) belongs the credit of attempting to establish it as a separate and distinct disease of the stomach. He gave to it the name *linitis plastica* (cirrhotic inflammation).

Of the terms offered, Brinton's baptismal name has won the most favor. This is especially true of the French and Italian literature. In his search for a distinctive title he suggests "that the inflammation of the filamentous network of areolar tissue, which seems to be the main characteristic of the lesion, might be well expressed by such a word as *linitis*" (from the Homeric *λίνον*, *ex lino factum*). He rejects the term *interstitial gastritis* "as the involvement of the mucous membrane is inconstant, late, and incidental"; *hypertrophy*, as it is an evident misnomer; *fibroid infiltration*, because it is not a true histological likeness.

Contemporary with Brinton the English observers, Handfield Jones, Wilks, Fox, Habershon, Hare, and Quain reported cases and considered the affection benign. For a considerable period following Brinton's observations, the view

that the lesion was benign met with the most favor. This was materially strengthened by the admirable articles of Hanot and Gombault (1882), Tourlet (1902). On the other hand, Garret (1892), Bret and Paviot (1894), Hoche (1903), Schacher (1903), forcibly declared the lesion to be malignant. This later conclusion dominates the French and Italian literature of to-day.

Opposed to this we have the operative findings and results of von Eiselberg (1902), Roux (1903), Sheldon (1904), Jonnesco and Grossman (1905), Deaver (1906), Lyle (1908), and the most excellent article of Kurt von Sury (1907).

Nature and Pathogenesis.—The pathogenesis of this affection is not yet clear, and it is impossible to say which of the many theories is correct. Brinton was well aware of the difficulty of making any definite statement, and warns that nothing short of "careful histories of cases during life and still more careful examinations of the corresponding specimens after death, in a far larger number than are at present at our disposal, can afford materials for any safe deductions." He recognizes the fact that a careful examination of the stomach may not reveal anything suggestive of cancer, yet the adjacent glands, lungs, liver, etc., may yield cancerous deposits.

Among the numerous theories that have been advanced are: that it is a stage of advanced gastritis; a primary atrophy (Fenwick); a result of chronic passive hyperæmia due to cardiac insufficiency (von Sury). Pedrizzini believes it to be a manifestation of senility; Huchard, an arteriosclerosis; and Poncet, Leriche, Villiers, and Phillippen claim that it has a tubercular element. Bouvert thinks it is due to a lymphatic obstruction; Wilks, that it is a primitive sclerosis of the submucosa; Garret and Bard, that it is a secondary sclerosis following a fusiform sarcoma. Bret and Paviot maintain that it is secondary to an infiltrating epithelioma. Shacher holds that it is an epithelioma pure and simple, starting from the mucosa, infiltrating the gastric walls, causing an irritative

connective-tissue hyperplasia, this irritation producing epithelial buds. Hoche maintains that it is an infiltrating epithelioma arising from an old cicatrizing ulcer. Danel's view is similar, but he places special stress on the cancerous involvement of the glands. Hanseman, in his work on endothelioma, says that many of the so-called scirrhus infiltrations of the stomach are endothelioma.

Delebet claims that linitis plastica is a diffuse cancer of the stomach. "The interest is not so much the question of the nature but how it advances. In linitis plastica secondary nests have been observed in all parts of the intestine; these could be attributed to primitive cell nests because they appear not in the mucosa, but in the cellular or muscular layers and others on the mesenteric side." They are due to a retrograde invasion of the lymphatics; they present a phenomenon identical to the one Handley has described in the breast under the name of permeation; this mechanism is not habitual, on the contrary it is only in these cases. The secondary nodules, which in ordinary cancers of the stomach are most often found in the liver, are lacking in a great majority of recorded cases.

In all these theories only two distinct ideas have been expressed: one, that linitis plastica is a special affection of an indefinite nature and cause; the other, that linitis plastica is a special form of scirrhus cancer.

Krompecher holds that gastro-intestinal sclerostenosis is not a mere disease of the pylorus, but is found in the intestines and peritoneum, and that it is the result of a chronic venous œdema caused by cardiac insufficiency and arteriosclerosis; and that the pathological process bears a close relation to scleroderma. (See Krompecher's conclusion, Case 70.)

Pathology.—Two forms are recognized, the local and the general. In the localized form we find indurated plaques on different portions of the viscus. This is rare. Viti's case is an example. The more common variety of this form is the one which is found in the pyloric region, forming a plaque of varying extent, often encircling and stenosing the pylorus.

Examples of this are the cases of Habersohn, Tilger, Boas, Chaput and Pillet, Brissaud, Oettenger and Tourlet. It is natural to suppose that the obstruction to the pylorus would lead to a dilatation of the stomach. This is seldom met with (exception, Nauwerk). The infiltration shades insensibly into the normal stomach tissue, but stops abruptly at the duodenum. An exception to this is Marcy and Griffith's case, where the infiltration extends to the duodenal entrance of the common duct. Herrenschmidt's case, in which the process has extended into the duodenum, pancreatic ducts, and common bile-duct, cannot be considered as an exception, for it is not a true linitis plastica.

In the generalized form the stomach may be normal in size, dilated, or contracted. Contraction is the rule, dilatation the exception. In a typical case we find a shrunken, thick-walled tube lying transversely across the epigastrium, suggesting by its size a segment of the large intestine. Often the walls of such a case are so rigid that if the stomach be removed it does not collapse but maintains its original shape. The peculiar dull grayish color of the peritoneum gives to the surface of the stomach a scarred looking appearance.

On section the stomach wall is greatly increased in thickness. Brinton says it may be six to eight times as thick as the normal wall. Despite the infiltration the layers remain distinct.

In the advanced cases all the coats are involved, the most marked involvement being in the submucosa, subserosa and serosa. According to Brinton the submucosa is ten to twenty times its normal thickness, the serosa and subserosa seven to ten times, the muscularis five to eight times, and the mucosa two to three times. The mucosa is often normal in appearance, "the secretory structures remaining substantially healthy," or it may be thickened and merged into the adjacent layers. Primitive atrophy is infrequent, ulceration rare. When there is marked involvement of the walls the knife fairly creaks as if it were cutting through cartilage. The cut section is comparatively bloodless, and no cancer juice can be

expressed. Tumor formation is uncommon; it is a flat infiltrating process. The lymphatic glands are small and hard, sometimes enlarged.

In the majority of cases evidences of an associated sub-acute or chronic peritonitis are prominent: lymph on the coils of the intestine, fibrous adhesions, ascites, thickening and opacities of lesser and greater omentum, white waxy-like plaques on the visceral and parietal peritoneum, thickening of the retroperitoneal tissue (retroperitoneal callus of Hanot and Gombault).

Analogous lesions are found in the colon, rectum, and small intestine. These lesions start from the mesenteric attachment and suggest a retrograde lymphatic involvement. These secondary involvements are considered by Bret and Paviot to be cancerous. Krompecher concludes that they are benign and suggests the similarity between the lesions and scleroderma.

Microscopical Examination.—The mucosa may be normal, or it may show evidences of a chronic productive or atrophic gastritis. In the advanced cases we meet with cystic dilatation of the tubules, constrictions, and other anomalies due to the pressure effects of the infiltrating connective tissue. This infiltration may lead to a sclerosis with atrophy, causing a complete disappearance of all the glandular elements (example, Osler's case).

The most marked and constant lesion found is a diffuse hypertrophy of the connective-tissue elements of the submucosa. The bundles of white connective tissues traverse the layers in irregular bands, surrounding the blood-vessels and interlacing with one another. The blood-vessels may show signs of endarteritis. The resemblance which this tangled interlacement of fibres bears to the weave of sail-cloth suggested to Brinton's mind the term "linitis."

It is a disputed question whether the epithelial cells found in the submucosa and muscularis are carcinomatous or whether they spring from the endothelium lining the normal lymph

spaces. The majority of observers consider these nests to be evidences of cancer. In the muscularis there is a combination of hypertrophy and atrophy of the muscle fibres. Bands of connective tissue are found passing from the submucosa through the muscularis to the subserosa. The subserosa shows a marked connective-tissue infiltration, the serosa a destruction of the endothelial cells. The lymphatic glands when involved show a chronic connective-tissue fibrosis.

In forming a mental picture of this lesion we are struck by the sharp contrast between the integrity of the mucosa with its absence of ulceration and the enormous thickening of the submucosa.

Etiology.—It is a disease of adult life. In Table A (true linitis plastica), the total number of cases in which the age is given is 64. Excluding three of these (Nos. 8, 55, 57) which cannot be properly admitted, the remaining 61 are grouped as follows:

Between 20-30 years	6 cases
Between 30-40 years	13 cases
Between 40-50 years	17 cases
Between 50-60 years	11 cases
Between 60-70 years	12 cases
Between 70-80 years	2 cases

In Table B (so-called malignant linitis plastica), the total number of cases in which the age is given is 54. Excluding Nos. 36 and 50, the remaining 52 are grouped as follows:

Between 20-30 years	6 cases
Between 30-40 years	9 cases
Between 40-50 years	10 cases
Between 50-60 years	12 cases
Between 60-70 years	11 cases
Between 70-80 years	4 cases

It is more often found in men than in women. In Table A (true linitis plastica), there are 41 men; 22 women. Sex not given in 15 cases.

In Table B (so-called malignant linitis plastica), there are 33 men; 19 women. Sex not given in 5 cases.

A considerable number of the cases give a history of cardiac or arterial trouble; a few give an alcoholic history. Syphilis is not a factor. Multiple peptic ulcers have been advanced as a cause. Poncet, Leriche, Villiers, and Phillippen believe that tuberculosis plays an important part. Occupation trauma was noted in the cases of Hare, Snellen, and Schacher. Ascites and peritonitis are results, not causes.

Symptoms.—There were no symptoms referable to the stomach in Viti's case, the antemortem diagnosis being chronic bronchitis and arteriosclerosis. Halipré's and Beaurain's second case gave a rheumatic history, their third case died of diabetes; leucoplakial plaques were found in the mouth. In Bouchard's and Kurt von Sury's cases the stomach symptoms were overshadowed by arteriosclerosis and cardiac complications. The diagnosis was not made until a few months before death. In the great majority of cases the onset is insidious; in a few abrupt. For months or years there is an indefinite history of dyspepsia, then definite progressive gastric symptoms appear, followed by severe anæmia, starvation, cachexia, and death.

In the first stage the symptoms are vague and inconstant, they are best described by the term indefinite dyspepsia. These symptoms gradually give way to a more or less constant anorexia, occasional vomiting, and an indistinct gastric pain and tenderness.

The involvement of the peritoneum brings its own train of symptoms according to the organs and structures involved; constipation, attacks of partial obstruction, and marked diarrhoea have been reported; of these diarrhoea is the most common. Ascites and œdema may come on early, but as a rule they are terminal symptoms.

A transverse sausage-shaped tumor or a sense of resistance was reported in 13 cases. The motility of the tumor depends on the amount of peritoneal involvement. Hæmatemesis is

not common; melena rare. An increase of HCl is uncommon, a decrease or absence of free HCl is the rule. Lactic acid is present in a small number of cases.

The vomiting, at first inconstant and inconsequent, becomes more frequent; towards the last it is incessant. If the lesion be near the cardia it simulates a regurgitation from an œsophageal stricture. The vomiting is never that of a dilated stomach, but of a small rigid organ which is intolerant to the quantity of food rather than the quality. The patients complain of a suffocative tightness in the epigastrium; this distressing symptom is partially relieved by vomiting. The stomach cannot be distended and will only hold small quantities of fluid.

Valuable information can be obtained from an X-ray examination. In Jonas's case the shadow showed a small stomach of the infantile type drawn well up under the ribs, the outline of the shadow was sharp, no peristaltic wave was noted. Pressure on the stomach wall forced the bismuth paste into intestine.

Course.—Unrelieved by surgical measures, the disease is uniformly fatal. In 43 cases of true linitis plastica, in which the duration of the symptoms was given, the shortest was 3 months, the longest 20 years, the average 49 months. In 37 cases of the so-called malignant type, the shortest duration was one month, the longest 15 years, the average 23.9 months.

Diagnosis.—The diagnosis of linitis plastica is rarely made during life; it comes to light as a surprise of the operating table or the autopsy room.

Boulton (1862) reports a case in which the diagnosis of linitis plastica was made during life. At autopsy the gross findings were those of linitis plastica, but as no microscopical examination was made, the honor of the first diagnosis which was proven by autopsy and microscopical examination goes to Deguy (1896); the credit of the second goes to Osler (1901).

An accurate clinical diagnosis between scirrhus cancer and linitis plastica is impossible. After the most careful, pains-

taking microscopical examinations failure has been recorded (Curtis's case). The points in favor of its benignity are: a history of cardiac insufficiency, general arteritis, pericarditis, etc.; its occurrence in younger people; its long duration; the absence of evidences of dilatation of the stomach; the slowly decreasing capacity of the stomach; the infrequency of hæmatemesis and melena; the character of the cachexia, it being a starvation rather than a poisoning. The size, distensibility, and the presence or absence of a pyloric stenosis can be determined by the X-ray and physical means. If a small shrunken stomach without pyloric tumor formation be found, the question of a linitis plastica might arise (Jonas's case).

The condition has been mistaken for (1) movable kidney, (2) movable spleen, (3) movable transverse colon, (4) portal cirrhosis, (5) pyloric stenosis with ulcer, (6) gastric ulcer with degenerative changes, (7) chronic peritonitis, (8) tubercular peritonitis. The diffuse infiltrating hypertrophic type of syphilis of the stomach could be readily mistaken for linitis plastica. Here we would depend on the history, the results of a course of anti-syphilis treatment, and the presence or absence of the Wassermann reaction.

Treatment.—The practical difficulties in making a diagnosis between this lesion and scirrhus cancer, and the possibility of it being a precancerous state, make gastrectomy the operation of choice provided the technical conditions are satisfactory. The condition of the patient, the presence of extensive adhesions, etc., may make gastro-enterostomy advisable. If gastro-enterostomy is impracticable, jejunostomy in Y gives a better clinical result than gastrostomy. Marchant and Domulin describe lesions similar to those of linitis plastica, in which the patients have been apparently cured by simple exploratory laparotomy and by gastro-enterostomy.

Total gastrectomy—three cases.

RIBERA (1895), reported by Boeckel. Total gastrectomy with œsophago-duodenal anastomosis; well four years after. This is the first operative case on record. It was a marked case of linitis plastica with numer-

ous adhesions between the stomach and the parietal peritoneum. At operation a portion of the abdominal wall was resected, as it was thought to be infiltrated with cancer. Microscopical examination proved it to be benign. (Table A, No. 53.)

VAN LEERSUM (1900). First performed pyloroplasty (Heineke-Mikulicz), no improvement; three months later did a total gastrectomy with œsophagojejunal anastomosis. (Table B, No. 38.)

JABOULAY (1904), reported by Gayet and Patel. Total gastrectomy; rectum removed one year later for a similar lesion. (Table B, No. 16.)

Partial gastrectomy—thirteen cases.

MONPROFIT (1898). Resection of one-third of the stomach; gastro-enterostomy in Y. (Table A, No. 28.)

DEBÉT (1900), reported by Brissaud. Pylorectomy; well four months later. (Table B, No. 10.)

VAUTRIN (1900). Partial gastrectomy; gastroduodenal anastomosis; died two and one-half years later of acute phthisis; no autopsy. (Table B, No. 17.)

WEISS (1900), reported by Schacher. Partial gastrectomy, two-thirds. Gastroduodenal anastomosis; well two and one-half years after. (Table B, No. 13.)

CHAPUT (1901), reported by Tourlet. First gastro-enterostomy; then pylorectomy; well ten months after. (Table A, No. 30.)

PONCET, reported by Mouriquard. Partial gastrectomy with gastro-enterostomy; fourteen days after the original operation the wound broke open, gastric fistula formed; spontaneous healing; well 22 months after. (Table B, No. 19.)

ROSSI (1903). Partial gastrectomy; patient died three months later from intestinal obstruction, the result of the operation. (Table B, No. 29.)

DURET (1904), reported by Danel. Partial gastrectomy with gastroduodenal anastomosis; died three days later from shock. (Table B, No. 45.)

TASINI (1906), reported by Morone. Partial gastrectomy; well 14 months later. (Table B, No. 43.)

QUENU (1906). Pylorectomy with posterior gastro-enterostomy. (Table B, No. 25.)

QUENU (1906). Extensive gastrectomy with posterior gastro-enterostomy; died three and one-half years after. (Table B, No. 26.)

LECENE (1908). Pylorectomy with gastro-enterostomy. (Table B, No. 24.)

TEMOIN (1910), reported by Roussy. Pylorectomy. No clinical details. (Table B, No. 59.)

Gastro-enterostomy—nine cases.

CHAPUT (1906), reported by Chaput and Pilliet. Gastro-enterostomy; died two days later. (Table A, No. 25.)

TRICONNI (1902), reported by Cignozzi. Gastro-enterostomy in Y; died eight days later from inanition. (Table B, No. 33.)

VAUTRIN (1903), reported by Vautrin and Hoche. Gastro-enterostomy; two months later an entero-anastomosis for vicious circle; died next day. (Table B, No. 11.)

SHELDON (1903). Anterior gastro-enterostomy; well at the present time (personal communication). (Table A, No. 46.)

ROUX (1904). Anterior gastro-enterostomy; well three and one-half years after. (Table A, No. 31.)

DEAVER (1906). Posterior gastro-enterostomy; died four years after; cause of death not known (personal communication). (Table A, No. 47.)

LYLE (1907). Posterior gastro-enterostomy; well at the present time. (Table A, No. 71.)

DERVEAU (1909). Posterior gastro-enterostomy impossible on account of adhesions. Anterior gastro-enterostomy. Died of inanition four months later. This was an extreme case of linitis plastica. Derveau says he should have done a jejunostomy in Y. (Table A, No. 58.)

Pyloroplasty—one case. See Van Leersum. (Table B, No. 38.)

Gastrostomy—two cases. (a) See Jonnesco and Grossman. (Table A, No. 34.) (b) Stretton (1909). Gastrostomy; died. (Table B, No. 27.)

Jejunostomy—four cases.

WICKERHAUSER (1902), reported by Cackvoic. Gastrectomy indicated but impossible; jejunostomy performed; died seven days later from peritonitis. (Table B, No. 34.)

CACKVOIC (1902). Jejunostomy; died seven days later from hypostatic pneumonia. (Table B, No. 35.)

V. EISELBERG (1902). Jejunostomy (Witzel). Well five years after. Eighteen days before this operation an exploratory laparotomy had been performed with the object of making a posterior gastro-enterostomy; this was found to be impossible. The abdomen was closed as the patient's consent for the formation of an artificial fistula had not been obtained. (Table A, No. 48.)

JONNESCO and GROSSMAN (1905). A typical gastrostomy was attempted under difficult conditions; it was not satisfactory, so a jejunostomy in Y was performed. Patient improved. A few weeks later an attempt was made to close the gastric fistula. Patient died two days later. (Table A, No. 34.)

Manual Dilatation.—FINNEY (1901), reported by McRae. Manual dilatation for hour-glass constriction; died four days later. (Table A, No. 36.)

CONCLUSIONS.

1. Diffuse fibrosis of the stomach occurs without cancer.
2. A large number of the cases reported are frankly cancer and have no claim to be termed linitis plastica. On the other hand, some of the cases reported as linitis plastica are

scirrhus cancers. "This is no doubt true of some of Brinton's cases, for at that period the microscopical examinations were of necessity inadequate." (Welch, personal communication.)

3. The clinical diagnosis is rarely possible and at the best is always problematic. The microscopical diagnosis necessitates a careful and prolonged search for nests of cancer cells in order to exclude scirrhus.

4. There is a possibility that the condition may be a pre-cancerous state, bearing somewhat the same relation to scirrhus cancer that gastric ulcer bears to gastric carcinoma.

5. The treatment is surgical.

TABLE A.—BENIGN.

Abstracts of Reported Cases of Linitis Plastica.

1. CARRIÈRE, male, age 52. Gastric symptoms for four months; no tumor felt. Autopsy; hypertrophy of the stomach most marked in the pyloric region but extending on lesser curvature and anterior wall. Microscopical: benign.

2. TROUSSEAU, male, age 25. Active gastric symptoms for three months; palpation gives a suggestion of thickened stomach wall. Autopsy: small, contracted, thick-walled stomach; no tumor. Diagnosis before autopsy: carcinoma. Microscopical: benign linitis plastica.

3. BALLET, female, age 59. For several months pain, vomiting, and tenderness; alcoholic history. Autopsy: ascites; stomach about size of transverse colon; surface smooth; no adhesions; no ulceration of the mucosa; tissue hard and grated under the knife. Microscopical: marked connective-tissue increase in the submucosa. Original diagnosis: carcinoma of the stomach; revised: benign linitis plastica.

4. MARIGNAC, female, age 29. For twenty years obstinate constipation alternating with diarrhoea; active gastric symptoms for six months; death from intestinal obstruction. Autopsy: ascites; stomach small, walls very thick, adhesions between stomach and colon; numerous small, white fibrous patches on the mesentery, peritoneum, and omentum; on rectum and colon contractions of fibrous tissue. Microscopical: lesions in the stomach, colon, and rectum similar; mucosa intact; submucosa four to five times normal thickness; muscularis hypertrophied; peritoneum thickened; seems to be a case of linitis plastica of the stomach, colon, and rectum.

5. NORNAGEL, male, age 23. Gastric disturbances for eight years; progressive pernicious anæmia. Autopsy: pernicious anæmia with small, thick-walled stomach; tissue grated under knife. Microscopical: suppression of the gastric glands and replacement by connective tissue; muscu-

laris and submucosa greatly thickened; glands non-malignant; case of pernicious anæmia with linitis plastica of the stomach.

6. BROCA, female, age 50. Gastric symptoms for seven years; pain and vomiting; no blood; tumor in the epigastrium. Autopsy: small thick-walled stomach. Microscopical: benign.

7. SNELLEN, male, age 52. Progressive gastric symptoms for two years; tumor in epigastrium. Autopsy: hypertrophy of the submucosa. Microscopical: mucosa shows lesions of chronic gastritis; submucosa compact, fibrous infiltration; hypertrophy of the muscularis with fibrous infiltration.

8. LEUDET, male. No clinical symptoms given. This case is quoted by Tourlet, but the evidence is not sufficient to call it a case of linitis plastica.

9. SCHOCH, male, age 32. Gastric symptoms for sixteen years. Autopsy: dilatation of the stomach with hypertrophy in the pylorus. Microscopical: marked thickening of the coats, most marked in the muscularis. This case is quoted by Tourlet, but seems to be a case of pyloric stenosis with dilatation of the stomach rather than a case of linitis plastica.

10. WILKS, female, age 44. Alcoholic; no previous history is stated; present history dates back several weeks; apparently died of peritonitis. Autopsy: ascites; chronic fibrous peritonitis with numerous adhesions; omentum and colon show marked thickening; stomach small, thick-walled, and rigid. Microscopical: hypertrophy of the muscularis with marked involvement of the submucosa; peritoneum dense and fibrous; transverse colon, sigmoid, and rectum showed a similar lesion to that of the stomach; no involvement of other abdominal organs.

11. HARE, female, age 33. Moderately alcoholic; gastric symptoms for four to five years; small tumor in the epigastrium. Autopsy: small, thick-walled stomach contained only four to five ounces; numerous adhesions; on section, hard, pearly gray in color; 1.5 to 2.5 cm. thick. Microscopical: no ulceration of the mucosa and all coats including the peritoneum showed connective-tissue changes. Diagnosis before autopsy was cancer of the spleen or of the peritoneum; revised diagnosis: linitis plastica. This patient was a laundress and was in the habit of pressing the iron against her stomach.

12. NAUWERK, female, age 23. No clinical symptoms given. Autopsy: stomach fills two-thirds of the abdominal cavity; at the pylorus there is a fibrous tumor formed by the hypertrophy of the walls of the stomach and duodenum; no ulceration; no peritonitis; no glandular involvement; thickness 14 mm. Microscopical: benign.

13. HENROT, male, age 59. Alcoholic; terminal symptoms were of three months' duration; loss of appetite, and diarrhœa; ascites, œdema of extremities; no tumor, epigastric pain, or vomiting. Autopsy: volume normal; both orifices contracted; walls very thick and resistant; no ulceration; fibrous plaques on small and large intestine; mesentery and omentum thickened. Microscopical: stomach and contracted portions of the small and large intestine showed the lesions of linitis plastica.

14. DURAND-FARDEL, reported by Letulle. Alcoholic; for eight months has presented classical symptoms of cirrhosis of the liver. Autopsy: stomach sausage-shaped; walls hard and thick; cut like cartilage; omentum shrunken and hard; liver cirrhotic. Microscopical: marked case of linitis plastica. Before the microscopical examination this was thought to be carcinoma of the stomach.

15. SCHMIDT, female, age 46. Alcoholic; gastric symptoms for eight years; vomited blood once; no tumor. Autopsy: ascites; diminution in size of all abdominal organs; stomach very small; walls hard and thick; no tumor, ulceration or scars. Microscopical: mucosa showed changes of the connective-tissue type; glands obliterated.

16. HANOT and GOMBAULT, male, age 45. For one year has complained of weakness and distention of the stomach; lately marked ascites and œdema of extremities; no tumor or vomiting. Autopsy: tuberculosis of the apices of the lungs; ascites; great and small omentum shrivelled up; adhesions between the pylorus, colon and neighboring structures; numerous glands; stomach small, thick walled; mucosa smooth and thick; lesions most marked in the region of the pylorus. Microscopical: non-malignant linitis plastica; glands chronic adenitis.

17. DUBUJADOUX, male, age 23. Non-alcoholic; for three months vague pains in the right hypochondrium; lately jaundice; some ascites and œdema around the ankles; no tumor. Autopsy: ascites; cirrhosis of the liver; stomach thickened at the pyloric end; mucous membrane pale and thick; marked increase in the submucosa and muscularis. Microscopical: benign linitis plastica.

18. FORMAND, male, age 38. For ten months has had dyspepsia and vomiting; lately the vomiting has assumed the characteristic of an œsophageal regurgitation; symptoms disappeared a short time before death; no hematemesis. Autopsy: stomach very small, walls hard, 2.5 cm. thick; volume 130 to 150 c.c. On the posterior wall there is an ulcer, the base of which is the spleen; the lesion was thought to be cancer, but on microscopical examination it proved to be benign linitis plastica.

19. KAHLDEN, female, age 60. Vague gastric trouble for one year and a half; vomitus contained blood during the last weeks; ascites; tumor. Autopsy: chronic fibrous peritonitis; atrophy and sclerosis of the omentum and stomach. Microscopical: diffuse connective tissue infiltration of all the walls of the stomach; the glands of the mucosa have largely disappeared.

20. VITI, male, age 68. Died with symptoms pointing to a chronic bronchitis and arteriosclerosis. Autopsy: small thick-walled stomach; no tumor; no ulcer; no stenosis of the pylorus. Microscopical: lesions of diffuse sclerosis throughout stomach; marked thickening of the mucosa; hypertrophy of the muscularis; serosa not involved.

21. ROSSINI, male, age 49. Gastric symptoms for fourteen years; vomiting for ten; no tenderness or tumor; no hematemesis. Autopsy: stomach, greater curvature reaches to the level of the umbilicus; mucosa grayish-white with black patches; a mild stenosis of the pylorus due to

hypertrophy. Microscopical: mucosa infiltrated with connective-tissue; glandular epithelium normal in some portions, absent in others, and in still others shows hyaline degeneration; some of the gland tubules are cystic. Clinical diagnosis before autopsy was chronic gastritis; stenosis of the pylorus with gastrectasis. A few days before death fibrous cancer of the pylorus. Linitis plastica.

22. ROSENHEIM, female, age 58. Symptoms for fifteen months; loss of appetite; pyrosis; nausea, tenderness, progressive emaciation, tumor and anachlorhydria, lactic acid present. Microscopical: showed a simple hypertrophy of the pyloric region; mucosa shows the lesion of an atropic gastritis. The clinical diagnosis before autopsy was pyloric cancer.

23. TILLET, male. Duration of symptoms not stated. Complaints of general weakness, dyspepsia, etc., without vomiting. Autopsy: stomach dilated; walls thick and cartilaginous, peritoneum not involved. Microscopical: hypertrophy of mucosa; connective-tissue infiltration of all the walls. Linitis plastica, benign.

24. GABBI, female, age 62. Gastric symptoms for several months; pain and vomiting acute in latter stages; anachlorhydria, lactic acid present; no blood; transverse tumor. Autopsy: ascites; grayish-white plaques resembling drops of wax on parietal and visceral peritoneum; stomach small; walls thick and hard; mucous membrane thrown into folds, pale; no ulceration; glands of the lesser curvature enlarged; similar thickenings in the intestine, most marked at the ileo-cæcal junction. Microscopical: hypertrophy of all the layers of the stomach, most marked at pylorus; in the different layers the maximum change is in the submucosa. Linitis plastica, benign. Microscopical examination of the intestinal lesions shows them to be benign.

25. PILLIET, male, age 55. Symptoms for about one year; lately, constipation and vomiting; hematemesis and melena; HCl present. Operation by Chaput. Thick walled, hypertrophied stomach; gastro-enterotomy; death 48 hours later. Autopsy: Microscopical: interglandular gastritis; marked involvement of muscularis and submucosa; linitis plastica; non-cancerous. (Cornil.)

26. DEGUY, male, age 57. Two previous attacks of acute articular rheumatism; gastric symptoms for over a year. The character of the symptoms led to the belief that this was an œsophageal condition; this, however, was ruled out by the use of a stomach tube; diffuse mass. Autopsy: small, thick walled, rigid stomach; no adhesions. Microscopical: linitis plastica, benign. (Cornil.) This is the first recorded case in which the diagnosis was made during life and sustained by microscopical examination after death.

27. PEDRAZZINI, male, age 66. Has been in the hospital a month; loss of appetite; pain in the epigastrium; transverse tumor. Autopsy: small, hard, thick-walled stomach; section cartilaginous; pearly gray in color; mucosa pale and anæmic; all coats involved; most noticeable in submucosa. Microscopical: linitis plastica, benign.

28. MONPROFIT, male, age 32. For two years pain, vomiting and constipation; movable tumor about the size of an egg. Operation: mass occupying one-third of the inferior portion of the stomach; resection of the stomach. Microscopical by Pilliet: Pseudo tumor composed of chronic inflammatory tissue, involving all the layers of the stomach.

29. HUCHARD, male, age 60. Alcoholic; chronic rheumatism; signs of atrophic cirrhosis with ascites; for six months aortic insufficiency. Diagnosis before autopsy: arteriosclerosis; stomach, sclerotic process involving all the walls; near the pylorus the lesion has a gross appearance of cancer. Microscopical: a simple sclerotic process.

30. TOURLET, from Ottenger's service, male, age 33. Gastric symptoms for eight years; progressive increase; no hematemesis; extreme constipation and diminished HCl; mass. Operation by Chaput: first, gastro-enterostomy; second, pylorotomy. Well ten months afterward. Gross and microscopical examination show it to be benign linitis plastica. Ottenger's opinion: inflammatory in nature and has chronic lymphatic oedema as its basis.

31. ROUX, male, age 33. Duration of gastric symptoms not given; felt full after meals; food did not seem to pass; later vomiting; no blood; no melena; anachlorhydria; transverse tumor. Operation: gastro-enterostomy by Roux. Hard, tube-like stomach; walls greatly thickened. Operation difficult due to adhesions and thickness of walls. Patient well three years after operation. Article excellently illustrated.

32. SMITH, female, age 34. Pain and vomiting; no tumor; small, hard, thick-walled stomach; greatest involvement region of the pylorus. Microscopical: linitis plastica, benign. Smith reports another case, but as no microscopic examination is given, it is not included.

33. A JACOBI, male, age 60. Gastric symptoms for more than six months; vomiting for six months. Diagnosis before autopsy was cancer of the stomach. Stomach was thick-walled and shrunken to the size of the small intestine; shows a progressive contraction of the stomach and gastric hypertrophy. Microscopical: benign. (Personal communication.)

34. JONNESCO AND GROSSMAN, male, age 42. Gastric symptoms for three years; pain, vomiting and tenderness; no tumor; at 52 cm. from the teeth stomach tube encounters an obstruction; stomach will only hold from 30 to 40 c.c. Operation: a typical gastrostomy attempted under difficult conditions due to the thick, hard walls; result unsatisfactory so jejunostomy performed; on account of a leakage an attempt was made to close the gastric fistula; the patient died two days after. Autopsy: shows the peritoneum free; no adhesions; stomach irregular ovoid in shape; capacity 40 c.c.; mucosa, no ulceration; section of the stomach grates under the knife and is pearly gray in color. Microscopical: enormous thickening of the submucosa; mucosa very little changed; muscularis infiltrated with connective tissue; serosa endothelium normal; connective-tissue in the submucosa. Linitis plastica, benign. Good article with excellent illustration.

35. v. SURY, male, age 60. Was treated for cardiac disease, then a diagnosis of carcinoma of the stomach and omentum with ascites was made. Had had stomach trouble for three years but the symptoms were overshadowed by the cardiac trouble until a short time before death. Autopsy: stomach, duodenum, colon, spleen, and liver bound together by firm adhesions; stomach small and thick walled. Microscopical: shows it to be a simple inflammatory lesion. (Schrumpmagen.) Excellent article.

36. McCRAE-OSLER, male, age 48. Gastric symptoms for five years; admitted to the hospital with a diagnosis of carcinoma of the stomach; free HCl; lactic acid present; can only take a small amount of nutrition at one time. Operation by Finney. Found a small, contracted stomach with an hour-glass constriction; manual dilatation of the constriction; died four days later. Autopsy: no signs of malignancy or ulceration. Microscopical diagnosis: cirrhosis of the stomach. This is the second case on record in which the diagnosis was made during life and confirmed by microscopical examination.

37. OSLER-HENRY, male, age 42. Alcoholic history; gastric symptoms for three years; pernicious anæmia. Autopsy: marked decrease in size of stomach; pylorus thickened; cicatrix of old ulcer on lesser curvature; especially noticeable is the atrophy of the mucous membrane and the marked hypertrophy of the muscularis mucosa.

38. FENWICK, male, age 45. Gastric symptoms for eighteen months. Autopsy: linitis plastica macroscopically and microscopically.

39. FENWICK, male, age 50. Gastric symptoms two to three years; cardiac insufficiency marked. Autopsy: linitis plastica.

40. FENWICK, male, age 54. Gastric symptoms for considerable period. Autopsy: linitis plastica.

(In going over these cases of Fenwick's they seem to have a marked likeness to the lesions of severe anæmia.)

41. JONES, male, age 66. Symptoms of anæmia and cardiac insufficiency. Autopsy: benign atrophy of the stomach.

42. GELHAM, male, age 45. Gradual progressive gastric symptoms. Autopsy: Microscopical: shows a degeneration and atrophy of the gastric tubules.

43. MARCY-GRIFFITH, male, age 46. Gastric symptoms for five years; alcoholic history; can only take small amounts of food; diarrhœa. Autopsy: stomach very small; lumen contracted; holds 60 c.c.; mucosa smooth; stomach wall 1.5 cm. thick. Microscopical: mucosa very little changed; marked connective-tissue overgrowth in the muscularis; stellate cicatrix in the mucosa.

44. CHARLES, male, age 73. Gastric symptoms, vomiting and diarrhœa. Autopsy: small, thick-walled, tube-like stomach. Microscopical: linitis plastica, benign. Illustrated.

45. OLLIVER, female, age 35. Gastric symptoms for two to three years. Autopsy: ascites; stomach small; holds 100 cm.; wall 1.5 cm. thick. Microscopical: linitis plastica, benign.

46. SHELTON, male, age 52. Gastric symptoms for fifteen years, gradually increasing; anæmia severe; no free HCl; no tumor; stomach will only hold a very small quantity. Before operation a probable diagnosis of benign stenosis was made; at the operation stomach was found to be greatly diminished in size, the walls thickened, firm, elastic and resistant; walls 1 cm. thick; anterior gastro-enterostomy; patient is well at the present date, May 11, 1911, eight years after operation.

47. DEEVER, male, age 46. Gastric symptoms for three years; no hematemesis; no melena; cannot retain more than 180 cm. at a time; no free HCl; no tumor. Operation: posterior gastro-enterostomy; stomach much contracted; walls dense and fibrous, resembling in appearance the shape of the small intestine; walls 2.5 cm. thick; died four years later, cause not known. (Personal communication.)

48. VON EISELBERG, female, age 41. Gastric symptoms for three years; feeling of pressure in stomach; anorexia and frequent vomiting; stomach's contents normal; stomach only held a small amount; pre-operative diagnosis: hour-glass stomach with adhesions. Operation: stomach bound down by adhesions; looked like a long, narrow sac; the surface of the stomach between the adhesions seemed to be covered by a net-work of scars; walls were that of a rigid tube; no ulceration; gastro-enterostomy was technically impossible and gastrostomy could not be performed as patient's consent had not been obtained for the formation of a fistula; eighteen days later a jejunostomy by the Witzel method was performed; patient is well five years after.

49. HADDEN, female, age 30. Gastric symptoms for ten months; Autopsy: stomach, tube-like, 10 cm. long; walls 1.2 cm. thick; mucous membrane roughened and ulcerated at the œsophageal end, remaining portion smooth and firm. Microscopical: mucosa has disappeared; great thickening of the submucosa; muscularis and serosa normal; no sign of malignancy.

50. TURNER, male, age 60. Gastric symptoms for one month; died three months later. Autopsy: two inches from the pylorus tight stricture; walls of the stomach greatly thickened and fibrous; ulcer on the anterior wall about the size of twenty-five-cent piece; mucosa traversed by fibrous tracts; numerous peritoneal adhesions. Author states that the extreme thickness was not due to the ulcer but the ulcer was the result of the condition.

51. LEITH (Albutt's system), case of diffuse cirrhosis of the stomach. Microscopical: benign.

52. ALBUTT (Leith), male, age 40. Ten years before had been treated for ulcer of the stomach. Autopsy: non-malignant cirrhosis of the stomach. Albutt believed that the process arose from the scar of a healed ulcer; judging from the scar the ulcers had been of considerable extent and severity, resulting in a keloid.

53. RIBERA, female, age 40. Gastric symptoms for five years; pain and vomiting; free fluid in the abdomen; tumor. Pre-operative diag-

nosis: cancer. Operation: total gastrectomy; small contracted stomach with adhesions; portion of abdomen wall resected as it was infiltrated. Microscopical: chronic inflammation with the predominance of connective-tissue element; no epithelial elements; old ulcer; well four years after; non-cancerous. First operative case on record.

54. HALIPRE-BEAURAIN, male, age 60. Diagnosis of diabetes; no gastric symptoms; leucoplakial patches in mouth and on lips. Autopsy: stomach small and thick-walled; glands enlarged. Microscopical: linitis plastica, benign; glands benign. This case died of diabetic cachexia. These authors reported two other cases which were cancerous in nature. See Table B.

55. JONAS (Van Orden), female, age 41. Had abdominal cramp from youth; lately pain and vomiting; no hematemesis; anachlorhydria; trace of blood in stools; resistance in the epigastrium; thought to be cancer. X-ray was taken and showed a stomach infantile in type; no stenosis; no peristaltic waves; bismuth passes readily into the small intestine; absence of stenosis speaks against cancer. This is the first reported case in which X-ray has revealed this condition.

56. BOULTON, female, age 22. Alcoholic; gastric symptoms for three years and a half; diarrhoea is marked; tumor. Made a diagnosis during life of linitis plastica (1862). On gross examination it appears to be linitis plastica, but as no microscopical examination is given, we cannot accept it. This is the first reported case in which the diagnosis was made during life.

57. HOLAND, female, age 35. Three years ago noticed an enlargement of abdomen; tumor; indefinite gastric symptoms. Autopsy: dense, fibrous adhesions involving viscera and omentum; small, thick-walled stomach. Microscopical: benign linitis plastica.

58. DERVEAU, male. Gastric symptoms for one year; pain, distress and vomiting; no hematemesis; anachlorhydria; cachexia; an extensive mass was felt and a pre-operative diagnosis of cancer of the pylorus with extension on the anterior wall and lesser curvature was made. Operation: stomach size of kidney and felt like kidney; numerous adhesions to all the neighboring organs; anterior gastro-enterostomy made; lumen of stomach greatly reduced; walls very thick. Case improved for a time but died four months later. Microscopical: extreme case of linitis plastica; benign.

59. PHILLEPPEN, female, age 36. History was that of ulcer of the stomach; gastric symptoms for eight years; had marked hypochlorhydria; diarrhoea toward the close; died in the hospital after two months' medical care. Autopsy: Macroscopical and microscopical: linitis plastica, benign; no ulceration. Writer thinks it is most often seen in tubercular subjects.

60-61. VIELLERS reports two cases of typical benign linitis plastica complicated by tuberculosis and states that this tubercular relationship is common in this disease of the stomach.

KROMPECHER reports on the anatomy, histology, and pathogenesis of nine cases of sclerotic stenosis of the stomach and intestinal tract.

62. Female, age 54. Death the result of a mitral stenosis. At autopsy a marked funnel-shaped stenosing hypertrophy was found; the pylorus was 7 cm. thick; on the surface of the stomach were fibrous patches; there was a similar lesion on sigmoid. The latter growth was the most marked at its mesenteric attachment. Microscopical: mucosa, nothing abnormal; muscularis mucosa, intact, œdematous; submucosa, sclerosis, hypertrophy and œdema with a characteristic obliteration of the capillaries; subserosa, peculiar calcareous deposit; circumscribed telangiectasis in the walls of stomach and sigmoid.

63. Male, age 47. Cause of death: purulent cystitis, pyelonephritis, amyloid degeneration of the kidney. There was an old fibrous pleurisy, fibrous peritonitis, an induration of spleen and pancreas and a marked funnel-shaped stenosing hypertrophy of the pylorus, dilatation of stomach and a circumscribed telangiectasis. Telangiectasis and fibrous patches in rectum. Marked hypertrophy and sclerosis of bladder and ureters. Microscopical: gastro-intestinal tract, bladder, and ureters showed a combination of hypertrophy, sclerosis and œdema with a tendency to the obliteration of the capillaries of submucosa.

64. Female, age 36. Diagnosis: cardiac insufficiency (bicuspid). Autopsy: marked arteriosclerosis, dilatation of heart, induration of lungs and liver, fibrous peritonitis, hypertrophy of pylorus and the small and large intestine, ulcer of rectum. Microscopical: marked necrosis of the atrophic mucous membrane of the small intestine, a combination of œdema, hypertrophy and sclerosis of the connective tissue elements, causing a compression of blood vessels which leads to an obliteration or dilatation.

65. Male, age 79. Chronic endocarditis of the mitral and aortic valves, arterio-sclerosis, empyema of lungs; hypertrophy and stenosis of pylorus; small intestine involved. Microscopical: œdema, sclerosis and hyalin degeneration of the thickened pylorus and corresponding lesion in the small intestine.

66. Female, age 64. Arteriosclerosis, secondary endocarditis, venous hyperæmia, œdema, pyloric hypertrophy. Microscopical: mild inflammation of mucous membrane; œdema, sclerosis and hyalin degeneration of the muscularis, submucosa and serosa.

67. Male, age 49. Arteriosclerosis, secondary endocarditis, marked pyloric hypertrophy, venous hyperæmia, œdema, and atrophy of intestine. Microscopical: mild inflammation of mucous membrane, œdema, sclerosis and hyalin degeneration of the muscularis, submucosa and serosa.

68. Male, age 62. Clinical diagnosis: gastric cancer. Autopsy: general arteriosclerosis, hypertrophy of heart; interstitial myocarditis, venous hyperæmia, œdema pylorus; marked hypertrophy with stenosis; intestine œdematous, at places atrophy. Microscopical: mild inflammation of mucous membrane, œdema, sclerosis and hyalin degeneration of the muscularis, submucosa and serosa.

69. Male, age 44. Villous cancer of bladder; fatty degeneration of heart muscle; adhesive pleurisy; œdema of lungs; fibrous perihepatitis; pyloric hypertrophy. Microscopical: marked œdematous involvement of the sclerotic and hypertrophic connective tissue of stomach and small intestine.

The above cases of Krompecher's series show a benign stenosis of the pylorus; with the exception of Case 68 they were all discovered at autopsy.

Cases 62, 65, 66, 67 had non-compensating hearts with arteriosclerosis. Cases 63 and 69 had bilateral adhesive pleuritis, empyema, perihepatitis and heart weakness. Cases 64 and 68 had extensive arteriosclerosis. Ages range from 36, 42, 49, 49, 54, 62, 64, 79—5 men, 3 women.

70. Male, age 22. Died of incarceration and peritonitis. Autopsy: degeneration of heart, œdema of lungs, œdema of small intestine; chronic peritonitis, wax-like plaques on the serosa of the stomach and intestine. These plaques show a combination of hypertrophy, sclerosis and œdema; some of them show calcareous sclerotic tissue. The sclerosis is most marked in the submucosa and many of the vessels are collapsed.

From the study of this case Krompecher pictures the steps: first, a chronic venous œdema caused by cardiac insufficiency, arteriosclerosis, callus pleuritis, chronic empyema, etc., and following on this is an œdema and induration. In the literature the reports, as a rule, are confined to the stomach and do not give the lesions of the other viscera. He concludes that gastro-intestinal sclerostenosis bears a near relation to venous congestion and œdema; third, the pathological process is similar to scleroderma, begins with œdema and ends with sclerosis.

LYLE. The patient, B. B., was transferred from the medical service of Dr. Janeway to the surgical service of Division B, St. Luke's Hospital.

Diagnosis.—Partial volvulus of stomach with abdominal adhesions.

Admitted June 11; transferred to surgical side, July 23, 1907.

History.—Woman, single, aged forty; Swede, occupation, cook.

Family History.—Father had kidney trouble and rheumatism. Three children had rheumatism, two had cardiac complications; third attack two years later, in bed five weeks; last January was in bed three weeks with an attack of rheumatism. This was followed by an otitis media, which lasted four weeks. Has nocturnal dyspnœa, suffers from pain, palpitation, dizziness, and syncope. Menses irregular and painful. No history of syphilis.

Present History.—Five days ago had sharp, cramp-like pains in the left lumbar and inguinal regions, radiating downward and inward into the thigh. These pains persisted for three days, then disappeared, to reappear on the second day after; felt nauseated, but did not vomit. These attacks came at irregular intervals and bore no relation to the taking of food. Complained of tenderness at a point just to the left of umbilicus.

Urination: Some increased frequency, somewhat cloudy; otherwise negative. Bowels constipated, appetite poor.

Physical Examination.—Patient, woman, weight 98 lbs.; large frame; poorly nourished; skin and mucous membranes anæmic; tongue clean.

Pulse: Irregular and of poor force.

Heart: Apex in sixth space, $4\frac{1}{2}$ inches to left; action somewhat irregular. There was a harsh, blowing, systolic murmur heard at the apex; a second one heard over the pulmonic area. Second pulmonic accentuated; right border one inch to the right.

Lungs: Clear.

Abdomen: Tenderness and slight muscular spasm one inch down and to the left of the umbilicus. No mass made out. Liver enlarged. Spleen not felt. Right kidney palpable. Rectal and vaginal examination negative.

Extremities: Superficial glands enlarged.

Blood: Leucocytes, 11,532; polynuclears, 74 per cent.; lymphocytes, 26 per cent.; hæmoglobin, 70 per cent. Urine normal.

Patient was examined in hot bath, and a sense of muscular resistance was experienced in the upper left quadrant.

Tuberculin injected. No reaction.

June 12: Patient still complained of severe attacks of pain in the epigastric region, had several attacks a day—no relation to the taking of food.

June 19: Attacks of pain continued—several every day. At times the pain radiated upward into the left axilla and across the abdomen. The attacks grew more severe.

Stomach analysis, June 4, 1907: Total acidity, 56; free HCl, 20; no lactic acid; no Boas-Oppler bacilli; no blood.

As the condition was steadily growing worse, she was trans-

ferred to the surgical side for operation, the diagnosis being gastric adhesions.

Operation by Dr. Lyle, July 27, 1907. On opening the abdominal cavity a mass of adhesions was encountered and the exact relation of the parts could not be made out. On separating the adhesions, it was found that the stomach was half rotated to the left, and the pylorus was firmly fixed by a short, dense, fibrous band to abdominal wall one inch and a quarter to the left of the umbilicus; there was a corresponding twist in the great omentum. The stomach itself was about half the size of a fist, puckered, and scarred. Over the region of the pylorus and lesser curvature the wall was markedly thickened and congested, suggesting a possible old ulcer or a new growth. A portion of tissue was removed for examination. Glands enlarged. It was decided to shorten the gastrohepatic ligament to prevent the recurrence of the rotation, and to do a gastro-enterostomy. During the shortening of the ligament, the patient's condition became serious, so the gastro-enterostomy was abandoned and the abdomen closed. The patient was returned to the ward. From the second day on she made an uninterrupted recovery. She left the hospital on the fifteenth day, apparently cured of all her symptoms.

The examination of the tissue removed showed granulation tissue, young connective tissue with areas of chronic inflammation. Nothing pointing to malignancy. Patient was seen eight months later, when she appeared to be well and strong.

Patient was again admitted to the hospital August 20, 1908.

History.—Since leaving the hospital she has been perfectly well, provided she limits her food in quantity. The quality does not seem to make any difference. Five weeks ago began to vomit half an hour after eating. The ingestion of food causes considerable pain, which pain is referred to the old scar. The patient can take only small quantities of food. Pain is sharp and cramp-like in character and radiates down the left leg to the knee; has gradually grown worse, and is very severe at the present time. Some burning in throat and eructations of gas. Has lost 25 lbs. in 8 months. Has grown quite weak. Occasional attacks of dizziness; no symptoms of loss of cardiac compensation. Stomach contents show a slight increase of total

acidity and free HCl on the first examination; the second a loss of acidity and free HCl.

Abdomen: Concave, soft, and relaxed. There is a firm linear scar, three inches long, half way between ensiform and umbilicus. Some tenderness in the epigastric region. No mass made out. Outline of stomach could not be mapped out. Six ounces of water distended the organ and gave considerable distress.

Diagnosis.—Possible gastric adhesions. With the previous history in view, it was thought advisable to place the patient under medical treatment for gastric ulcer before proceeding to operation. Patient put on Lenhartz's regimen. There was no improvement.

Operation September 9, 1908. Dr. Lyle.

On opening the abdominal cavity, a small, shrunken, and scarred stomach was found covered with adhesions—some of these dense, others thin; they extended from the anterior surface to the abdominal wall and from the lesser curvature to the under surface of the liver. The lesser curvature was represented by a dense, scar-like mass, radiating from which were folds of thickened tissue and adhesions. The distance from the pylorus to the œsophageal opening, measured along what remained of the lesser curvature, was less than one-half inch. The stomach was contracted to about the size of a goose egg, and was almost circular in shape. The stomach wall seemed to be twice the normal thickness.

A no-loop posterior gastrojejunostomy was performed; considerable difficulty was experienced, due to the smallness of the stomach and the thickness of its walls. The patient made a prompt and rapid recovery and was out of bed on the thirteenth day.

Since leaving the hospital she has been under careful observation, and is well and strong. Has gained 35 lbs., and worked steadily since leaving the hospital. The stomach contents still show a diminished acidity.

At the first operation the condition was not recognized, and it was thought to be a case of ulcer of the stomach with the formation of numerous perigastric adhesions, accompanied by accidental rotation of the stomach. The walls of the stomach at

that time were thickened and scarred. The freeing of the adhesions and shortening of the gastrohepatic ligament gave relief for almost a year.

At the second operation the uniform increased thickness of the walls and the marked diminution of the size of the stomach, plus the scarring, showing that something else than a contraction following a simple ulcer had to be dealt with. In the causation of this case we have a combination of two factors, each of which have been given as a cause of the disease. We have a well-marked history of chronic passive hyperæmia from cardiac insufficiency (the result of repeated attacks of rheumatism), and a strong possibility of multiple peptic ulcers. The chronic condition, plus the irritation set up by the ulcers, would undoubtedly be a sufficient stimulus to start changes in the connective-tissue elements of the stomach, and thus lead to marked general contraction; with the contractions would come of necessity a thickening of the walls, until we find a rigid organ with greatly diminished lumen and thickened walls, as found in this case.

NOTE.—Since the preparation of this report the patient has had four distinct attacks of toxic erythema, due to the taking of meat. These attacks can be controlled by the addition of some dilute HCl and pepsin, showing that her stomach digestion is below par for digestion of meats.

TABLE B.

Cases Reported as Linitis Plastica which are Malignant in Nature.

1. WEIL. T. G., male, age 61. Two months before entering hospital noticed a tumor in pyloric region; tenderness; regurgitating vomiting; died of purulent pleurisy. Cachexia was a very prominent symptom. Autopsy: stomach wall is 2.5 cm. thick; mucosa normal; submucosa was replaced by a hard, dry tissue which furnished no juice on scraping; head of pancreas and prevertebral glands invaded; peritoneum not involved. Microscopical: mucosa a little invaded by new growth; muscularis destroyed; submucosa makes two-thirds of the thickness; considers this a new plastic growth similar to a fusiform sarcoma.

2. LYONNET, female. Symptoms for ten months. Autopsy: enormous diffuse thickening of the whole stomach; secondary deposits in the pancreas and heart; ascites; enlargement of the glands. Microscopical: malignant linitis plastica.

3. BEZANÇON, male, age 41. Alcoholic; symptoms for a year; vomit-

ing quarter of an hour after eating; pain. Autopsy: stenosis of the pylorus formed of sclerotic tissue; no ulceration; stomach somewhat dilated. Microscopical: a typical epithelioma (Bezançon). Chronic inflammation without cancer. (Cornil.)

4. TILGER, female, age 35. Progressive gastric symptoms for two years; induration of pylorus; stomach dilated. Autopsy: marked stenosis of the pylorus; stomach walls thick. Microscopical: all coats hypertrophied; submucosa most marked; between connective-tissue there are masses of epithelial cells denoting cancerous degeneration; mucosa shows the lesions of chronic gastritis. (Plates with detailed examination.)

5. BRET-PAVIOT, female, age 43. Alcoholic; insidious onset beginning four years before; last eighteen months has had attacks of colic and diarrhoea; persistent vomiting; no hematemesis; no melena; stomach tube appeared to stop at the cardia; tumor. Autopsy: ascites; stomach white in color, cylindrical with the calibre of the colon, two-thirds of its extent involved; numerous adhesions; similar lesion in colon; two tumors on ovary, possible cancers or fibro-miomata. Microscopical: all coats show connective-tissue infiltration, the maximum being in the submucosa; epithelial cells are found here and there. Examination of an ulcer in the cardiac region shows it to be non-cancerous. This is classed by Tourlet as benign but it is undoubtedly malignant.

6. LEPINÉ, cit. by Bret-Paviot, female, age 49. Progressive gastric symptoms for two years; diarrhoea and vomiting at close; no hematemesis; no melena. Autopsy: small, thick-walled, adherent stomach; section is pearly white in color and grates under knife; glands of the lesser curvature involved. Microscopical: mucosa lymphoid hyperplasia; submucosa infiltrated with undulating connective-tissue fibres in the meshes of which are epithelial cells. This is quoted by Tourlet as being benign but is undoubtedly malignant.

7. PILLIET-SAKORRAPHIS, male, age 31. Pain in the right side; loss of appetite; ascites; thought to be a cirrhosis or tubercular peritonitis; patient never vomited; died three years and a half after onset. Autopsy: stomach about the size of the closed fist; walls thick and hard; numerous adhesions; mucosa thickened; no ulceration. Microscopical: all coats of the stomach equally involved; embryonal infiltration at the pylorus; at greater curvature the mucous membrane is thinned; numerous round and fusiform cells present; the muscularis mucosa has disappeared; submucosa connective-tissue, infiltration with fusiform cells; vessels show endarteritis; the colon presents similar lesions. Tourlet classifies this as a benign case.

8. PETIBON, male, age 54. Has had indefinite gastric symptoms for years; symptoms of chronic gastritis for six months; tumor. Autopsy: small, thick-walled stomach; oesophagus involved; glands enlarged. Microscopical: linitis plastica, cancerous.

9. VERHOEGHE, male, age 60. Gastric symptoms for more than a year; regurgitation is marked; alcoholic. Autopsy: shrunken, rigid stomach

resembling coil of small intestine. Microscopical: cylindrical epithelioma with connective-tissue infiltration: linitis plastica, malignant.

10. BRISSAUD (Tourlet), male, age 34. Alcoholic; lues six years previously; gastric symptoms for a year; vomits immediately after taking food; lately vomiting has become incessant; stomach is not dilated; tumor in the region of the pylorus; no hematemesis; no melena. Operation: pylorotomy by Debét. Pre-operative diagnosis: chronic gastritis with fibrous stenosis of the pylorus. Portion removed at operation shows a neoplasm in the form of a cylinder 6 cm. long; consistency of the walls recall a fibroma or myoma. Microscopical: by Monod. This is quoted as benign by Tourlet. Schacher says it is a typical epithelioma. P. 77. Patient well four months after.

11. VAUTRIN-HOCHE, male, age 29. Gastro-enterostomy; two months after operation patient returned, suffering from a vicious circle; entero-enterostomy performed; death the following day. Stomach small, thick walled; retroperitoneal adhesions. Microscopical: diffuse epithelioma appearing in the zone of an old ulcer.

12. SCHACHER, service of Brissaud, female, age 55. Active gastric symptoms for five months; patient had had pains in the epigastrium for six years but had attributed them to her occupation which necessitated pressing with a heavy iron; constipation is marked; she had a total abdominal hysterectomy performed; both ovaries showed cysts. Operation gave no relief. Autopsy: stomach presents a hard, rigid, annular thickening; numerous adhesions; colon adherent to stomach, gall-bladder invaded by new growth; stomach adherent to posterior abdominal wall; cæcum involved at the base of appendix; sigmoid and rectum also show cancerous invasion. Microscopical: shows it to be a gastric tumor localized in the lesser portion of the stomach with anterior and posterior peritoneal adhesions. Schacher considers this a linitis plastica of cancerous nature. The course of the tumor has terminated in a generalization of the disease showing that the linitis plastica was a malignant cancer of epithelial origin. Excellent article, well illustrated.

13. WEISS (Schacher-Boechel), female, age 50. Gastric symptoms for several years; tumor. Operation: partial gastrectomy. Weiss thought it to be pure linitis plastica. Microscopical examination was made by Hoché. There is some doubt about the diagnosis of linitis plastica as an epithelial infiltration was found at the site of the old ulcer. This appears to be a diffuse infiltration of typical epithelial elements in the sclerotic inflammation of the cicatrix of an old ulcer; possibly this is a cancer developing on an old ulcer(?). This case is reported by Boechel as being well two years and a half after. (Boechel, p. 162, No. 38.)

14. SPILLMANN (Schacher), male, age 47. Gastric symptoms for one year; stomach not enlarged; no tumor; death from cachexia. Autopsy: lesions of linitis plastica most marked in pyloric region. Microscopical: diffuse neoplastic infiltration of an epithelial type in the sclerotic tissue.

15. WEISS, male, age 32. Died shortly after admission to hospital of

apparent cancer of the stomach; pylorus admitted little finger; antrum occupied by a tumor about the size of half a mandarin; stomach wall hard, pearly white in color; retroperitoneal "callus"; mesenteric and duodenal plaques. This is a case of epithelial linitis plastica type, having its origin in an old ulcer.

16. GAYET-PATEL, female, age 44. Gastric symptoms for eight months; no dilatation; no stasis; diffuse tumefaction in the region of the pylorus; free HCl absent. Total gastrectomy by Jaboulay. Stomach movable; pylorus hard and thickened; mottling and cloudiness of peritoneum; in the mucous membrane, near the pylorus, is a small erosion. Microscopical: evidence of connective-tissue in all layers; here and there cells of an epithelial character. Probably inflammation started from a cancerous base. One year later rectum was removed; it showed a recurrence. (Bret-Paviot.)

17. VAUTRIN, male, age 29. Gastric symptoms; vomiting; no hematemesis; pyloric tumor; partial gastrectomy. Patient died two years and a half after of acute tuberculosis; there were no signs of a recurrence. At the first examination two years and a half before there were no cancerous elements; a later examination showed suspicious cells in the connective tissue stroma indicating an epithelial degeneration. Boechel, p. 95. Schacher says it was linitis plastica of epithelial type. Final conclusion—scirrhus epithelioma of glandular origin.

18. CURTIS showed the stomach, ileum and colon as examples of linitis plastica, benign. On account of the protest he went over the sections again and found a small area which was cancerous. (Neurofibromatose carcinomateus.) "There exists in this case a very systemic propagation of the cancer cells following the tracks of the nerves." No clinical symptoms given.

19. MOURIGUAND, male, age 28. Gastric symptoms for eighteen months; tumor at the pylorus; stasis; free HCl; partial gastrectomy. (Poncet.) Well twenty-two months after. Diagnosis of linitis plastica was made (Paviot); there were signs of an old ulcer. Microscopical: hypertrophy of glandular region; glands non-cancerous; infiltration of submucosa and muscularis by a new production of connective tissue. Paviot considers these cells to be of neoplastic nature.

20. MOUISSET-CHALIER, male, age 71. Gastric symptoms for two years; pain on pressure; sense of resistance in epigastrium; no hematemesis. Autopsy: small, rigid, tube-like stomach; numerous peritoneal adhesions; cut section pearly white; no ulceration in mucous membrane; no tumor; cancerous mass in liver. Microscopical: linitis plastica, cancerous.

21. MÉNARD, female, age 53. Gastric symptoms for two years simulating a stenosis of the œsophagus; smooth tumor in the epigastrium; intestinal paresis; cachexial death. Autopsy: cancerous infiltration of the stomach, omentum and mesentery. This does not seem to be a case of linitis plastica but rather a case of carcinoma of the stomach.

22. FAROG, male, age 41. Vague gastric symptoms for one year; clinical course that of cancer. Autopsy: cancerous linitis plastica of the stomach;

neoplasm starting in the mucous membrane of the pylorus and extending to the abdominal walls. Patient had a neoplasm in the breast. No diagnosis is given other than that there were nodules in the skin. This looks like a recurrence after a breast tumor and not linitis plastica.

23. LORRAIN, male, age 55. Gives the symptoms of pyloric obstruction. Autopsy: linitis plastica, cancerous.

24. LECENÈ, male, age 50. Presents cancer of the pylorus with stenosis; stomach intolerant of food; anachlorhydria. Operation: pylorotomy; stomach small, rigid and tube-like. Microscopical: carcinomatous cells found in the mucosa and in the neighboring lymphatic; involvement most marked in submucosa and muscularis.

25. QUENU, male, age 32. Gastric symptoms for four years; transverse mass in the epigastrium. Diagnosis: carcinoma of the pylorus. Operation: partial gastrectomy. Gross findings those of linitis plastica. Microscopical: shows it to be cancerous in nature.

26. QUENU, female, age 28. Gastric symptoms for less than a year; epigastric tumor. Pylorotomy. No adhesions. Gross: had the aspect of scirrhus cancer. Microscopical: linitis plastica, malignant. Died three years and a half after.

27. STRETTON, female, age 56. Symptoms for three months which were supposed to be due to a cancer of the œsophagus. Gastrostomy was done. Stomach was hard, thick walled and contracted; capacity about one and a half ounces. (Leather bottle stomach.) Microscopical: shows it to be a spheroidal cell carcinoma.

28. ROSSI, male, age 50. Clinical diagnosis cirrhosis of the liver. Autopsy: Gross: appearance is that of linitis plastica with adhesions; stomach is small and drawn up under the ribs; perisplenitis and perihepatitis. Microscopical: mucous membrane shows evidence of an old tubercular inflammation in the muscularis mucosa; the submucosa shows a great enlargement due to connective-tissue in which are found some cellular fusiform elements of epithelial nature. He does not consider them cancerous; gland parenchyma not affected; capillaries invaded by epithelial elements similar to those of the gastric wall. This appears to be linitis plastica of a cancerous nature. Note: *possible tubercular influence*.

29. ROSSI, female, age 20. Symptoms for five months; tumor; no free HCl, blood or lactic acid. Operation: partial gastrectomy. Gross: linitis plastica; no ulceration; cylindrical, thick-walled sac; glands enlarged. Died three months later of intestinal obstruction. Microscopical: linitis plastica, cancerous.

30. TESTI-ZACCARIA, male, age 50. Gastric symptoms for ten months; alcoholic; vomiting; had characteristics of cancer. Gross: linitis plastica. Microscopical: scirrhus carcinoma; nature could not be accurately determined. The author has no doubt that this is an epithelial form of a scirrhus cancer.

31. CARDI, female, age 32. Gastric symptoms for two years; vomiting; HCl absent. Microscopical: linitis plastica without adhesions; lesions occupy pyloric half; on surface are white, fibrous plaques. There is a

possibility of old ulcer. Microscopical: high grade of linitis plastica with scirrhus cancer.

32. CIGNOZZI, male, age 61. Clinical diagnosis: hepatic cirrhosis with ascites; no direct gastric symptoms; intense diarrhoea; ascites and œdema of lower extremities. Autopsy: ascites; thickened posterior wall; waxy looking deposits on parietal peritoneum; thickened gastric wall with nodules; nodule in the lung; heart showed myocarditis with cardiac insufficiency; chronic tuberculosis of right pulmonary apex. Cause of death, acute perforating peritonitis. This seems to be a case of scirrhus cancer having its origin in the glandular elements. Cignozzi classes this case as a primary retroperitoneal endothelioma with secondary nodules in the stomach and left lung.

33. CIGNOZZI, female, age 38. Gastric symptoms for fifteen years; dilated stomach; tumor in the pyloric region. Operation: Gastro-enterostomy (Triconni); pylorus the seat of an annular tumefaction which extends from the pylorus to the posterior wall and greater curvature. Died eight days later. "Probable linitis plastica of endothelial type. The form of the cells, the arrangement of the alveoli and tubules, the absence of any epithelial elements exclude a cancerous process and point to an endothelial neoplasm."

34. CACKOVIC, male, age 36. Lung symptoms for two years; gastric symptoms of long standing; stomach held 100 c.c.; no tumor. Operation: jejunostomy. (Wickerhauser.) Gross: typical linitis plastica. Gastrectomy indicated but technically impossible so jejunostomy was done. Died from peritonitis seven days later. Microscopical: showed it to be cancerous.

35. CACKOVIC, male, age 60. Gastric symptoms for six months; sense of resistance at pylorus; stomach holds 50 c.c. Operation: stomach small, thick walled; presents a tumor about the size of an apple; numerous adhesions. Jejunostomy. Patient died seven days later from hypostatic pneumonia. Diagnosis: linitis plastica, malignant.

The diagnosis in the above two cases was made during life. In reading them over they seem to be nothing more than scirrhus cancers.

36. SCOTT reports a case non-malignant stenosis of the pylorus and duodenum; the pathological findings make it doubtful whether this case should be admitted.

37. POLLOCK, female, age 49. Gastric symptoms for two years; sense of resistance in the pyloric region. Autopsy: shrunken stomach, thick walled; thought to be cancer but careful examination shows no ulceration or malignancy; there seemed to be some doubt in Quain's mind so it is placed in the malignant list.

38. VAN LEERSUM-ROLGENS, female, age 22. Gastric symptoms for years; anachlorhydria. Operation: marked stenosis of the pyloric region, extending into the duodenum; pyloroplasty failure; three weeks later gastrectomy. Gross: showed specimen of linitis plastica arising from an old ulcer. Microscopical: epithelial elements found; malignant.

39. FOLLI-BERNARDELLI, male, age 60. Gastric symptoms for ten

months; no hematemesis. Autopsy: Gross: linitis plastica. Microscopical: linitis plastica, cancerous type; involvement of glands.

40. FOLLI-BERNARDELLI, male, age 62. No clinical history. Autopsy: Gross: linitis plastica; no ulceration. Microscopical: linitis plastica, cancerous.

41. FOLLI-BERNARDELLI, male, age 47. Gastric symptoms for one month; vomiting with small traces of blood; tumor in the epigastrium; ascites. Autopsy: very small, thick-walled stomach adherent to diaphragm and pancreas; mucosa pale; no ulceration. Microscopical: submucosa greatly thickened; contained abundant cellular elements; the epithelial cells had coalesced, forming small centres and infiltrating the neighboring tissue; glands enlarged; linitis plastica, cancerous.

42. FOLLI-BERNARDELLI, male, age 79. No clinical history; complete involvement of the stomach which was shrunken, thick walled and cartilaginous to feel; mucous membrane smooth; no ulceration; glands involved. Microscopical: marked increase in connective-tissue elements with infiltration of epithelial cells. Linitis plastica, cancerous.

43. MORONE, female, age 46. Gastric symptoms for eighteen months; tumor in the epigastrium; marked anæmia; hypochlorhydria; hematemesis. Pre-operative diagnosis: cancer of the lesser curvature developing on an old ulcer with secondary involvement of the pylorus. Operation by Tasini. Partial resection; linitis plastica of half the stomach; glands not involved. Patient well fourteen months after. Microscopical: linitis plastica (epithelioma).

44. RIVET, male, age 52. Gastric symptoms for two months; alcoholic; no hæmatemesis; no melena; no tumor. Autopsy: tuberculosis of the lungs; stomach contracted and adherent. Microscopical: linitis plastica, cancerous. The interest in this case is that there were no symptoms until two months before death.

45. DANIEL, female, age 34. Gastric symptoms for two years; alternating diarrhoea and constipation; no distention; no definite hematemesis or melena; marked cachexia; transverse cylindrical tumor. Diagnosis: epithelioma of the pylorus extending to the greater curvature. Operation: partial gastrectomy (Duret). Patient died three days later of shock. Autopsy: stomach small, shrunken and thick walled; omentum thickened and covered with whitish plaques; glands enlarged; no adhesions. Microscopical: linitis plastica, cancerous.

46. DUPLANT, male, age 73. No clinical symptoms. Autopsy: showed a greatly shrunken stomach covered by a thickened serosa; the thickened pylorus gave the appearance of a neoplasm; it is a diffuse neoplastic infiltration of the stomach with ulceration of the mucous membrane; glands enlarged. From gross examination the diagnosis lies between benign linitis plastica and a diffuse epithelioma. Microscopical: sections were not given at the time of the report.

47. MARIE. No clinical history; no data as regards autopsy. Statement is made that it is linitis plastica of a cancerous type.

48. MARIE. No clinical history. Autopsy: small, hard stomach with a retroperitoneal callus; no visible tumor; small ulcer near the pylorus; linitis plastica, cancerous.

49. LORRAIN, male, age 55. In the hospital for a short time with symptoms of stenosis of the pylorus. Autopsy: small, thick walled, contracted stomach. Microscopical: linitis plastica, cancerous.

50. BABES-MIRONESCA describe a case in which the gross findings somewhat resemble a linitis plastica but the microscopical examination shows it to be a gelatinous carcinoma.

51. HALIPRÉ-BEAURAIN, male, age 54. Alcoholic; gastric symptoms for eight months; no solid food tolerated; small tumor. Autopsy: stomach shrunken; feels like a rubber cylinder; middle of the transverse colon shows a fibrous ring; fibrous patches on the small intestine; glands enlarged. Microscopical: linitis plastica, cancerous.

52. HALIPRÉ-BEAURAIN, male, age 72. Rheumatic; no gastric symptoms; diarrhœa. Autopsy: stomach resembles a segment of the small intestine; greater curvature measures 6 cm.; walls very thick and resistant. Microscopical: linitis plastica, malignant. There were no gastric symptoms in spite of the advanced pathological condition. The patient died of bronchial pneumonia.

53. DELAMARE-BRELET, male, age 63. Alcoholic; gastric symptoms for four years; ascites and œdema of the lower extremities; alternating diarrhœa and constipation; the symptoms seem to be those of cirrhosis. Autopsy: ascites; liver small; mass of adhesions involving gall-bladder, greater and lesser omentum, etc.; stomach small, thick walled; holds only 150 cm.; 10 to 15 mm. thick; glands hard and small. Linitis plastica of an epitheliomatous nature.

54. FAROY, female, age 41. A few indefinite gastric symptoms before entering hospital; vomiting and progressive cachexia; death three months and a half after entering. Autopsy: small, contracted, thick-walled stomach; circular infiltration extending from the greater to the lesser curvature and making an hour-glass contraction; small intestine involved; suspicious nodules in the skin; numerous adhesions to the neighboring organs.

55. COVA-BONS, age 64. Died at 70. Symptoms recurred several times. First diagnosis was diffuse carcinoma of the stomach; second, linitis plastica with pyloric stenosis(?). Sense of resistance in the epigastrium; anachlorhydria; lactic acid present; cachexia marked. Autopsy: linitis plastica, cancerous.

56. ROUSSY presents before the Anat. Soc. of Paris four specimens of linitis plastica, in which the cancerous nature is evident from the histological examination. Two are of the generalized form and two of the localized.

Female, age 61. Linitis plastica, generalized. Short time in hospital and died of profound cachexia. No diagnosis was made. Stomach small,

hard and retracted under the liver; marked thickening of the coats; lesser and greater omentum involved; large and small intestine not involved; no secondary deposits of the viscera; lymphatic glands hard. Microscopical: grayish white; no ulceration. Histological examination shows it to be linitis plastica, cancerous.

57. Male, age 65. Linitis plastica, generalized. Stomach small, thick walled; 2 cm. thick at pylorus; large part of the transverse colon involved, 10 mm. thick; numerous adhesions; no ulceration of mucous membrane; no ascites. Microscopical: linitis plastica, cancerous; perivascular sclerosis marked in the submucosa.

58. Linitis plastica, localized. Cancerous; limited to the pyloric region. Pre-operative diagnosis: stenosis of the pylorus. Operation: gastrostomy. Small, thick-walled stomach; no evident involvement of the peritoneum. Microscopical: linitis plastica, cancerous.

59. Linitis plastica, localized. Limited to the pylorus. Operation: pylorotomy by Temoin of Bourges. Small, thick-walled stomach; pylorus 28 mm. thick; mucous membrane healthy. Microscopical: linitis plastica, cancerous.

60. LENORE-COURCOUX described a case under the title of "septicémie cancéreuse secondaire" in which the examination of the local lesion in the stomach suggested a linitis plastica of a cancerous type.

BIBLIOGRAPHY.

- ¹ Allbutt, C.: Cit. by Leith. System of Medicine, p. 467.
- ² Andral: Clinique médicale, t. ii, p. 59.
- ³ Andral: Précis d'anatomie pathologique, 1835, t. i.
- ⁴ Ascoli, V.: Sulla linitis plastica del Brinton; Osservazioni critiche a proposito di un recente lavaro. Policlinico-Medica, 1st Octobre, 1894.
- ⁵ Babes-Mironescu: Plastische Linitis und Magenkrebs. Ref. Münch. med. Woch., 1907, No. 32, p. 1605.
- ⁶ Baginsky, A.: Über normalen und pathologischen Befund des Lymphgefäßendothels kindlichen Darmwand. Zentrbl. f. med. Wissensch, 1882, No. 4.
- ⁷ Ballet: Observation de gastrite chronique, avec rétraction de l'estomac, et épaississement énorme des tuniques musculaire et celluleuse. Bull. Soc. anat., de Paris, 1877, p. 644.
- ⁸ Bandein: Hypertrophie considérable de la muqueuse du cardia et de la grande courbure de l'estomac. Bull. Soc. anat., de Paris, 1837, vol. xxxii, p. 245.
- ⁹ Bard: Anatomie générale des tumeurs.
- ¹⁰ Bard: Archives de physiologie, 1885.
- ¹¹ Bennett, H.: Cancerous and Cancroid Growths. London, 1849.
- ¹² Bensaude et Okingzo: Retrecissement cancéreux multiplis de la partie sous-diaphragmatique du tube digestif. Arch. de Med. Exp. et d'Anat. Path., 4 juillet 1906.

- ¹³ Bensaude et Rivet: Les cancers multiplis du tube digestif. *Gaz. des Hôpitaux*, Sept. 28, 1907.
- ¹⁴ Bérault: Hypertrophie chronique de l'estomac simulant un cancer. *Bull. Soc. anat.*, 1847, p. 10.
- ¹⁵ Bernabei: *Boll. d. sez. d. culture d. scienze med. n. r. Accademia d. fisiocritia di Siena.*, vol. v, 1807.
- ¹⁶ Bezançon, F.: Rétrécissement cancéreux du pylore (pas de cancer pour Cornil). *Bull. Soc. anat.*, 1892, p. 802.
- ¹⁷ Boas. Über hypertrophische Pylorus Stenose (stenosirende Gastritis) und deren Behandlung. *Archiv. f. Verdauungskrankheiten*, 1898, Bd. iv, 47-62 et 368.
- ¹⁸ Boeckel, J.: De l'ablation de l'estomac. Paris, 1903.
- ¹⁹ Bouveret, L.: *Traité des maladies de l'estomac*, Paris, 1893.
- ²⁰ Boyer: Hypertrophie de la membrane musculeuse de l'estomac. *Bull. Soc. anat.*, 1848, p. 49.
- ²¹ Brandt: Die Stenose des Pylorus, *Inaug. Dis. Erlangen*, 1851, S. 33.
- ²² Brellet, M.: La linite plastique de Brinton. *Gaz. des Hôpitaux*, Paris, 1905, p. 1599-1605.
- ²³ Bret et Paviot: Linite plastique. *Revue de méd.*, 1894, p. 384.
- ²⁴ Bricheteau: *Cliniques de l'hôpital Necker*.
- ²⁵ Bricheteau: Affection squirreuse de l'œsophagus du pylore, difference entre cette maladie et le cancer. Thèse Paris.
- ²⁶ Brinton: *Diseases of the Stomach*, 1864.
- ²⁷ Brissaud: Linite Plastique; pylorectomie; guérison, *Semaine méd.*, 1900, p. 415.
- ²⁸ Broca: Hypertrophie du pylore. *Bull. Soc. anat.*, 1850, p. 207.
- ²⁹ Bruch: Über Magenkrebs und Hypertrophie der Magenhäute in anatomischer oder klinischer Hinsicht. *Zeitschrift für ration. Med.*, 1849, Bd. viii.
- ³⁰ Budd: *On the Organic Disease and Functional Disorders of the Stomach*, London, 1855.
- ³¹ Bullett: Obliteration of the Stomach as a Result of Gastric Ulcer. Duodenostomy. *ANN. SURG., Phila.*, 1907, xiv, p. 30-33.
- ³² v. Cackovic, M.: Über totale Verkleinerung. (Schrumpfung) des Magens und über Jejunostomie. *Archiv. f. klin. Chir.*, 1902, p. 409.
- ³³ Cardi: Sulla linite plastica del Brinton. *Policlinico sez. méd.*, 1896, p. 576.
- ³⁴ Carrière: Hypertrophie partielle des parois de l'estomac. *Bull. Soc. anat.*, 1863, p. 163.
- ³⁵ Carswell: *Path. Anat.*, London, 1838, Tab. i-ii.
- ³⁶ Carter: *Diseases of the Stomach. Med. Fortnightly*, St. Louis, xix, 61-70, 1901.
- ³⁷ Chalvet: Hypertrophie concentrique de l'estomac. *Bull. Soc. anat.*, 1859, p. 68.
- ³⁸ Chaput et Pilliet: Linite hypertrophique non cancéreuse. *Bull. Soc. anat.*, 1896, p. 154.
- ³⁹ Chaput et Pilliet: *Observation clinique*, id., p. 22.

- ⁴⁰ Chaput et Oettinger: Présentation de notre malade. Bull. et mém., Soc. de chir., 3 juillet 1901, p. 794.
- ⁴¹ Chardel: Monographie de dégénération schirrheuse de l'estomac. Paris, 1908.
- ⁴² Charles, J.: An Account of Cirrhosis or Fibroid Infiltration of the Stomach. Dublin Jour. of Med. Sci., 1875, vol. lix, p. 201.
- ⁴³ Charrot-Bouchard-Brissaud: Traité de Médecine, Part III.
- ⁴⁴ Chiari: Über Magensyphlei Internat. Beitrag. zur. Wissenschaf Med. (Virchow Festschr.), 1891, ii, p. 297.
- ⁴⁵ Chifoliau et Masson: Linite Plastique. Soc. anat., Paris, 11 Novembre, 1910.
- ⁴⁶ Cignozzi, O.: Dui casi di endothelioma dello stomaco. La Ref. Med., 1905, pp. 17, 18, 19, 20.
- ⁴⁷ Colonna, G.: Le stenosi piloriche non maligne. Gaz. med. di Torino, 1900, li, p. 267.
- ⁴⁸ Cornil: Rapport sur l'observation Carrière. Bull. Soc. anat., 1864, p. 238.
- ⁴⁹ Cornil et Ranvier: Manuel d'histologie pathologique, 1884, t. ii.
- ⁵⁰ Cova, A., et Bons, G.: Contributo allo studio clinico anatomico ed istologico della linite plastica di Brinton. Gazz. d. osp., Milano, 1909, xxx, 569-571.
- ⁵¹ Coyon et Legros: Les stenoses pyloriques. Revue générale in Gazette des Hôpitaux, 1898, p. 917.
- ⁵² Cruveilhier: Anatomie pathologique, 1835, t. iii, p. 25.
- ⁵³ Curtis, F.: Étude sur un cas de linite plastique gastro-intestinale ou péritonito nodulaire sclérogène stenosante. Archiv. de Med. exper. (1908), also Bull. et mem. de la Soc. anat., 1909, No. 1, p. 14.
- ⁵⁴ Curtis, F.: Nouvel examen du cas linite plastique présente dans la seance du 16 janvier 1909. Bull. et mém. Soc. anat. de Paris, 1909, No. 3.
- ⁵⁵ Damaschino: Leçons sur des maladies des voies digestives, 1888, p. 497.
- ⁵⁶ Danel: La linite plastique. localisée. Jour. de Sci. Méd. de Lille, janvier 1904, p. 4904.
- ⁵⁷ Dawosky, S.: Hypertrophie du tissu cellulaire sous-muqueux du pylore chez un enfant de dix semaines. Archiv. générales de méd., 1843, p. 93.
- ⁵⁸ Deaver-Ashurst: Surgery of Upper Abdomen. Phila., 1908, vol. i, p. 234-237.
- ⁵⁹ Debove et Rémond: Traité des maladies de l'estomac, p. 222.
- ⁶⁰ Deguy: Un cas de linite plastique. Bull. Soc. anat., 1896, p. 314.
- ⁶¹ Delamare-Brelet: Soc. Méd. des Hôpitaux. Feb. 19, 1909. Linite plastique de l'estomac et de la Vesiculæ Billaire. Also Tribune Méd. Paris, 1909, t. xli, p. 166. Bull. et mém. Soc. anat., 1909, lxxxiv, p. 166-169.
- ⁶² D'Espine et Picot: Traité des maladies de l'enfance. Edition de 1900, p. 631.

- ⁸⁰ Doyen: *Traité chirurgical des maladies de l'estomac*, 1895.
- ⁸¹ Dubey: *Gaz. Hebdomen*, 1883, p. 108.
- ⁸² Dubujadoux: Sur une variété de cirrhose encore inédite accompagnant la gastrite chronique avec sclérose sous-muqueuse hypertrophique. *Gaz. hebdomadaire de méd. et de chir.*, 1883, No. 12, p. 198.
- ⁸³ Duclos: Tumeur fibro-plastique de la petite courbure de l'estomac. *Bull. Soc. anat.*, 1854, p. 200.
- ⁸⁴ Duplant: Epithelioma diffus de l'estomac ou linite plastique. *Soc. nat. de Méd. de Lyon. Lyon Med.*, 1899, No. 10, p. 337.
- ⁸⁵ Durand-Fardel: *Bull. Soc. anat.*, 1879, p. 305.
- ⁸⁶ Einhorn: *Diseases of the Stomach*, p. 165.
- ⁸⁷ Einhorn: Atrophy of the Stomach. *N. Y. Med. Record*, June 11, 1893, p. 650.
- ⁸⁸ Einhorn: Diagnosis and Treatment of Pyloric Stenosis. *Med. Record*, Jan. 10, 1895.
- ⁸⁹ v. Eiselberg: A Case of Linitis Plastica of the Stomach (Brinton) Cured by Jejunostomy. *Surg., Gyn. and Obs.*, 1908, vol. vii, p. 253-7.
- ⁹⁰ Eppinger: Totale Scirrhus des Magens (Totale Magenschumpfung). Cit. by v. Cackovic.
- ⁹¹ Ewald: *Chronik der Verdauungskrankheiten*, 1893, vol. ii, p. 187.
- ⁹² Faggi: Case of Diffused Suppurative Inflammation of the Stomach. *Trans. Path. Soc., London*, 1875, vol. xxvi, p. 81.
- ⁹³ Farog: Linite plastique cancéreuse. *Bull. et mém. de la Soc. anat.*, 19 Feb., 1909, No. 2.
- ⁹⁴ Fauvel: *Bull. Soc. anat.*, 1858, p. 224.
- ⁹⁵ Fenwick: On Atrophy of the Stomach and the Nervous Affection of the Digestive Organ, London, 1880.
- ⁹⁶ Folli-Bernardelli: Sulla linite plastica del Brinton. *Ref. Med.*, 1902, Nos. 279, 280, 281, 282.
- ⁹⁷ Formad: Chronic Hypertrophic Cirrhosis of the Stomach with Gastric Ulcer and Colloid Change of Mucous Membrane. *Jour. Am. Assoc.*, 1887, p. 599.
- ⁹⁸ Fox, W.: Hypertrophy of the Walls of the Stomach. *Reynolds Encyclopedia*, 1872.
- ⁹⁹ Fox, W.: *The Diseases of the Stomach*. London, 1872, p. 209.
- ¹⁰⁰ Frenoy: Des Faux cancers de l'estomac. Thèse Paris, 1896.
- ¹⁰¹ Gabbi, U.: Su di un caso di linite plastica (gastrite del Brinton). *Ref. Med.*, 1893, 6 et 7 Sept., vol. iii, Nos. 56 et 57.
- ¹⁰² Galliard: Les cirrhosis de l'intestin. *Méd. Moderne*, Jan., 1897.
- ¹⁰³ Garret, J.: Contribution à l'étude des néoplasmes de l'estomac; du cancer conjonctif sous-muqueux. Thèse de Lyon, 1892.
- ¹⁰⁴ Gärtner: Über den diffusen Scirrhus des Magens und seine Beziehung sur sog. gutartigen Magenverhärtung. *Inaug. Dis. Tübingen*, 1878. Cit. by v. Sury.
- ¹⁰⁵ Gayet-Patel: Un cas de gastrectomie totale pour linite plastique. *Archiv. générales de méd.*, 1904. No. 13.
- ¹⁰⁶ Gluge: *Atlas der patholog. Anatomie*, Jena, 1850, ix. Lieferung.

- ⁹² Guichard, O.: Des rétrécissements intrinsèques et non cancéreux du pylore, anatomie pathologique et diagnostic. Thèse Paris, 1874.
- ⁹³ Guelliot, O.: Faux cancer du pylore. Union médicale du Nord-Est, 1900, xxiv, p. 261-265.
- ⁹⁴ Habershon: Pathological and Clinical Observations Respecting Morbid Conditions of the Stomach, London, 1857, p. 99.
- ⁹⁵ Habershon: Diseases of the Stomach. London, 1869.
- ⁹⁶ Hadden, W.: A Case of Extreme Contraction of the Stomach. Trans. of Path. Soc., London, 1891.
- ⁹⁷ Halipré-Beaurain: Linite plastique cancéreuse et non cancéreuse. Revue Méd. de Normandie. Rouen, 1906, vol. vii, p. 401-413.
- ⁹⁸ Handley, W.: Cancer of the Breast and its Operative Treatment.
- ⁹⁹ Hanot et Gombault: Étude sur la gastrite chronique avec sclérose sous-muqueuse hypertrophique et rétro-péritonite calleuse. Archiv. générales de physiologie, 1882, t. ix, p. 410.
- ¹⁰⁰ Hare: Transaction of the Path. Soc. Med., 1853, t. iv, p. 129.
- ¹⁰¹ Hartmann: Rapport sur une observation de Mauclaire: Ulcère du pylore formant tumeur. Bull. et mém. Soc. de chir., 1899, p. 481.
- ¹⁰² Hayem et Lion: Article: Estomac, Traité de médecine et de thérapeutique, 1896, t. iv, p. 462.
- ¹⁰³ Hemmeter: Diseases of the Stomach. Phila., 1897.
- ¹⁰⁴ Henrot: Transformation fibreuse de la tunique musculaire de l'estomac, d'une partie du petit et du gros intestin. Gaz. des Hôpitaux, 1878, p. 975 et Union médicale et scientifique du Nord-Est, 1878, p. 11.
- ¹⁰⁵ Herrenschmidt: Linite cancéreuse gastro-duodenale. Bull. Soc. anat., 1908, p. 542.
- ¹⁰⁶ Hirsch: Freie Vereinigung der Chirurgen, 22 juin, 1896.
- ¹⁰⁷ Hoché: Étude sur la linite plastique. Rev. de Méd., 1903, pp. 1079-1116.
- ¹⁰⁸ Holland: Case of Fibrosis of the Stomach and Peritoneum. Med. Press, London, 1889.
- ¹⁰⁹ Horn: Einiges über Magencirrhose. Inaug. Dis. Kiel, 1869. Schriften du Universitatkiel. Cit. by v. Sury.
- ¹¹⁰ Huchard: Traité des maladies du cœur et de l'aorte, 1899, p. 132.
- ¹¹¹ Huchard: Des faux cancers de l'estomac. Bull. méd., 1894, p. 243.
- ¹¹² Hunt, G.: Trans. of the Path. Soc. of London, 1897-98.
- ¹¹³ Jaboulay: Lyon méd., 1905, p. 396.
- ¹¹⁴ Jacobi, A.: Progressive Contraction of the Stomach with Gastric Hypertrophy. N. Y. Med. Record, vol. xvii, 1880, p. 730.
- ¹¹⁵ Jonas: Zur Diagnostik der Schrumpfmagens. Wien. med. Woch., 1909, lix, pp. 260-263.
- ¹¹⁶ Jones, Hanfield: Path. and Clin. Obs. Respecting Morbid Conditions of the Stomach, London, 1854.
- ¹¹⁷ Jones, Hanfield: Diseases of the Stomach, London, 1855, p. 121.
- ¹¹⁸ Jonnesco-Grossman: Contribution à l'étude de la linite plastique. Rev. Chir., 1909, pp. 18-32.
- ¹¹⁹ Journal des Praticiens, 1897, Nos. 3. La Linite Plastique.
- ¹²⁰ Kahlden: Über chronische sclerosirende Gastritis. Centralbl. f. klin. Med., 16 Avril, 1887, p. 281.

- ¹³⁹ Kammerer, F.: Benign Obstruction of the Pylorus. Tr. Am. Surg. Assn., Phila., 1900, xviii, p. 74-96.
- ¹⁴⁰ Karewiski: Über einen Fall von Chlorzin Kvergiftung nebst Bemerkungen Jejunostomie. Berlin klin. Woch., S. 1896, No. 50.
- ¹⁴¹ Kehr: Zur Chirurgie der Gallenblasenkrankheiten. Berlin klin. Woch., 1893, No. 2, p. 39.
- ¹⁴² Krompecher, E.: Zur Anatomie, Histologie und Pathogenese der gastrischen und gastro-intestinal Sclerostenose. Zeigler Beitrag, vol. xlix, 1910, 2 Heft., p. 384.
- ¹⁴³ Kussmaul: Über die Behandlung der Magenerweiterung mittelst der Magenpumpe. Deut. Archiv. f. klin. Med., 1869, vi, p. 455.
- ¹⁴⁴ Labadei-Lagrange et Deguy: Les Perviscerthes. Arch. Générales de Med., 1898, vol. ii, p. 385.
- ¹⁴⁵ Landerer: Über angeborene Stenose des Pylorus. Inaug. Dis. Freiburg, 1879. Cit. v. Sury.
- ¹⁴⁶ Lange, J.: Über stenosirende pylorus hypertrophie Sauglingsalter. Münch. med. Woch., 1901, xlviii, 280-282.
- ¹⁴⁷ Lebert: Die Krankheiten des Magens, Tübingen, 1878, S. 525.
- ¹⁴⁸ Lebert: Anatomie pathologique, t. ii, p. 301.
- ¹⁴⁹ Lecène: Un cas de linite plastique de l'antre pylorique. Bull. et mém. de la Soc. anat., 1908, No. 10, p. 540.
- ¹⁵⁰ Leith: Cirrhosis of the Stomach, Albutt System of Med., vol. iii, p. 440, London, 1900.
- ¹⁵¹ Le Noir-Courcoux: Septicémie cancéreuse Secondaire. La Presse Med., No. 21, 1908. No. 94, p. 757-58.
- ¹⁵² Lesser: Cirrhosis ventriculi. Inaug. Dis., Berlin, 1876.
- ¹⁵³ Letulle: Un cas de gastrite chronique accompagnée de sclérose sous-muqueuse et péritonéale. (rapport sur une observation publiée en 1879 par Durand-Fardel). Bull. Soc. anat., 1883, t. lviii, p. 495.
- ¹⁵⁴ Letulle: Diagnostic du cancer de l'estomac. Presse médicale, 15 juillet, 1896.
- ¹⁵⁵ Leube: Die krankheiten des Magens und Darm. Handbuch der spec. Pathologie u. Therapie. (Ziemessen), 1876, vol. vii, 2.
- ¹⁵⁶ Leudet: Hypertrophie simple des parois de l'estomac. Bull. Soc. anat., 1853, t. xxviii, p. 145.
- ¹⁵⁷ Leudet: Bull. Soc. anat., 1852, p. 128, paragraph 16.
- ¹⁵⁸ Leuk: Untersuchungen sur pathologeschen Anatomie des Menschlichen Magens, Zeitsch. f. klin. Med., Band xxxvii, p. 296.
- ¹⁵⁹ Lieutaud, J.: Historia anatomico-medica, Paris (ed. Portal), 1767.
- ¹⁶⁰ Lieutaud, J.: Synopsis of the Universal Practice of Medicine, Trans. by Atlee, Philadelphia, 1816.
- ¹⁶¹ Luton: Dictionnaire de méd. et de chirurgie pratiques, t. xxiv, p. 150; t. xiv, p. 201 et 270.
- ¹⁶² Lorrain: Linite plastique cancéreuse. Bull. et mém. de la Soc. anat., 1909, No. 1, p. 43.
- ¹⁶³ Lyonnet: Linites cancéreuses, ganglions de Troisier, ascite chyleuse, généralisation au cœur. Lyon méd., 28 mars, 1897, p. 453.

- ³⁴⁴ Macquet: Rapport sur l'observation de Boyer. Bull. Soc. anat., 1848, p. 49.
- ³⁴⁵ Maier: Beiträge zur angeborenen Syphilis Stenose, Virchow's Archiv., 1895, cii, S. 413.
- ³⁴⁶ Maranger, O.: Contribution à l'étude de l'atrophie de l'estomac. Thèse Nancy, 1882.
- ³⁴⁷ Marchant-Gérard: Rapport sur une observation de M. Demoulin; Rétrécissement inflammatoire du cæcum. Bull. et mém. Soc. de Chirurgie, 1899, p. 651.
- ³⁴⁸ Marchant, Gérard et Demoulin: Sur les tumeurs et rétrécissements inflammatoires de la région pylorique de l'estomac et du segment iléocæcal de l'intestin. Revue de gynécologie et de chirurgie abdominale, 1899, p. 894.
- ³⁴⁹ Marcy et Griffith: On Muscular Hypertrophy of the Stomach. Am. Jour. of Med. Sci., 1884.
- ³⁵⁰ Marie, R.: Linite plastique. Bull. et mém. de la Soc. anat., 1908, p. 535.
- ³⁵¹ Marignac de: Rétrécissement par hypertrophie des tuniques cellulaires et musculaires du rectum et du côlon transverse. Epaissement analogue de l'estomac. Bull. Soc. anat., 1877, p. 519.
- ³⁵² Martin, C. F.: Modern Medicine, Osler, 1908, vol. iv, p. 311-319.
- ³⁵³ Mathieu, A.: Traité des maladies de l'estomac et de l'intestin, 1901, p. 713.
- ³⁵⁴ Mathieu, A.: In Traité de médecine. Charcot, Bouchard, Brissaud, t. iii.
- ³⁵⁵ McCrae: Cirrhosis of the Stomach. Johns Hopkins Bull., Jan., 1901, p. 21.
- ³⁵⁶ Ménard: Infiltration cancéreuse totale de l'estomac des épiploons et du mésentère (linite cancéreuse). Bull. et mém. de la Soc. anat., 1909, No. 1, p. 23.
- ³⁵⁷ Meinel, H.: Untersuchungen über die sogenannte gutarlege Pylorus Hypertrophie und Scirrhus des Magen. Ziegler's Beitrag., 1902, vol. xxxi, p. 479.
- ³⁵⁸ Mikulicz u. Kausch: Über Magen carcinoma. Handbuch des Praktischen Chirurgie. von Bergman, von Bruns u. von Mikulicz, vol. iii.
- ³⁵⁹ Monard: Jour. Am. Med. Soc., 1902.
- ³⁶⁰ Monneret: Affection organique de l'estomac, du foie et du péritoine. Gaz. des Hôpitaux, 1841, p. 469.
- ³⁶¹ Monprofit: Gastrectomie et gastro-entérostomie. Bull. de la Soc. de Chir., mars 1898, p. 282.
- ³⁶² Moorhead: Combined Cardiac and Pyloric Stenosis. The Practitioner, June, 1911, vol. lxxxvi, p. 831.
- ³⁶³ Morone: Contribution à l'étude de la linite plastique basée sur une observation clinique. La Reforma. Med., 1908, Nos. 21, 22, 23.
- ³⁶⁴ Mouisset-Chalier: Linite gastrique compliquées de cancer secondaire du Foie. Lyon Méd., 1909, p. 1004.
- ³⁶⁵ Moulin, M.: Lancet, June, 1907.

- ¹⁸⁸ Mouriquand: La linite plastique du pylore. Lyon Méd., 1907, p. 473.
- ¹⁸⁹ Müller, J.: Demonstration eines Falles von Schrumpfmagen. Sitzungsbericht der Physio-Medic. Gesellschaft, Würzburg, 1902, p. 11. Cit. by v. Sury.
- ¹⁹⁰ Nauwerck: Ein Fall von hypertrophischer Stenose des Pylorus mit Hochgradiger Magenerweiterung. Deutsch. Archiv. f. klin. Medicin, 1878, xxi, S. 574.
- ¹⁹¹ Nicoll: Congenital Hypertrophic Stenosis of Pylorus; with an Account of a Case Successfully Treated by Operation. British Med. Jour., Sept. 1, 1900.
- ¹⁹² Nothnagel: Cirrotische Verkleinerung des Magens und Schwund der Labdrüsen. Deutsch. Archiv. f. klin. Medicin, Bd. 24, 1879, p. 352.
- ¹⁹³ Oehler: Ein Fall von totaler Magenschrumpfung. Inaug. Dis. Leipzig, 1905. Cit. by v. Sury.
- ¹⁹⁴ Oettinger: De la stenose hypertrophique du pylore (linite plastique à localisation pylorique; maladie fibroïde du pylore; gastrite hypertrophique stenosante). La Semaine Méd., No. 19, p. 153, 1902.
- ¹⁹⁵ Oliver: Case of Fibroid Induration of the Stomach with Chronic Peritonitis and Ascites. Edinburgh Med. Jour., March, 1891.
- ¹⁹⁶ Olivier et Halipré: Gastrite scléreuse hypertrophique de nature cancéreuse (linite plastique de Brinton). Normandie méd., 1 avril, 1896.
- ¹⁹⁷ Orth, J.: Präkarzinomatöse Krankheiten und künstliche Krebs. (Zeitsch. f. Krebsforschung.), Bd. x, p. 42.
- ¹⁹⁸ Osler, Henry: Atrophy of the Stomach with the Clinical Features of Progressive Anæmia. Amer. Jour. Med. Sci., xc, 1886, p. 498.
- ¹⁹⁹ Pedrazzini, F.: Gastrite ipertrofica sottomucosa (linite plastica di Brinton). Gaz. med. lomb., Milano, 1898, lvii, p. 65.
- ²⁰⁰ Petibon: Contribution à l'étude de la gastrite scléreuse hypertrophique; ses rapports avec le cancer. Thèse de Paris, 1895, decembre.
- ²⁰¹ Pilliet: Cas de sclérose sous-muqueuse avec hypertrophie musculaire de la portion pylorique de l'estomac. Bull. Soc. anat., 1889, p. 538.
- ²⁰² Pilliet et Sakorraphos: Gastrite sous-muqueuse hypertrophique avec rétro-péritonite calleuse. Bull. Soc. anat., 1892, p. 288.
- ²⁰³ Pilliet: Observation d'érosion avec cirrhose gastrique. Bull. Soc. anat., 1891, p. 703.
- ²⁰⁴ Pohl, J.: Handb. d. path. Anat., 1804, vol. ii.
- ²⁰⁵ Pollock: Chronic Thickening of the Stomach and Omentum, Somewhat Resembling Malignant Disease. Trans. of Path. Soc., London, 1853, vol. iv, p. 129.
- ²⁰⁶ Poncet-Leriche: Bull. Acad. Med., 1905, p. 53.
- ²⁰⁷ Quain: Trans. of the Path. Soc. of London, 1870, xxi, p. 168.
- ²⁰⁸ Quenu: Deux cas de pylorotomie pour linite plastique; de la dilatation de l'œsophagus à la suite d'une gastrectomie presque totale. Bull. et mém. de la Soc. Chir., Paris, 1906, No. 27, pp. 731-40.
- ²⁰⁹ Rakowak, L.: Ein Fall von Gastritis Submucosa. Wiener. Med. Presse, 1874, No. 25.

- ¹⁸⁹ Ramoino-Morgagni: Le cancer infiltré de l'estomac simulant la linite plastique, janvier, 1899. Résumé in *Revue générale de pathologie interne*, 1899, 20 avril, p. 115.
- ¹⁹⁰ Rendu: Leçons de clinique médicale, 1890, t. ii, p. 10.
- ¹⁹¹ Ribera, J.: *Revesta de Medecin a. y. Cirugia*. May and June, 1905, Nos. 602-604. Also Boechel observation, xxviii.
- ¹⁹² Riegel: *Cirrhosis Ventriculi*. Diseases of the Stomach. Nothnagel's Encyclopedia, American Ed., p. 497.
- ¹⁹³ Rivet: *La Linite Plastique*. La Clinique. Paris, vol. v, 1910, pp. 193-7.
- ¹⁹⁴ Rivet: Linite cancéreuse avec retro-péritonite calleuse et métastasis multiplis. *Soc. anat.*, Paris, 1905.
- ¹⁹⁵ Robin, Ch.: *Clinique de Trousseau*, t. iii, p. 74, edit. 1873.
- ¹⁹⁶ Robin, A.: *Traité des maladies de l'estomac*, 1901.
- ¹⁹⁷ Robson (Mayo): *Keen's Surgery*, vol. iii, p. 825.
- ¹⁹⁸ Rokitsansky: *Lehrbuch der pathologischen Anatomie*, 1861.
- ¹⁹⁹ Rosenheim: Société de médecine berlinoise, séance du 11 juillet, 1894. Cit. by Tourlet.
- ²⁰⁰ Rossi: Linite plastica e carcinoma infiltrato dello-stomaco. *La Clin. Med.*, 1905.
- ²⁰¹ Rossoni: Un caso d'inflamazione cirrotica dello-stomaco con atrofia glandolare. *Lo Sperimentale*, 1883, pp. 500-511.
- ²⁰² Roussy, G.: Quatre cas de linite plastique de Brinton. *Bull. de l'Assn. Français pour l'étude cancer*. Vol. iii, Nos. 8, 9, pp. 455-461, Dec., 1910.
- ²⁰³ Roux: Linite plastique san néoplasme. *Rev. Méd. Sci.*, 1905, No. 1, p. 53.
- ²⁰⁴ Roux, J. Ch.: *La Linite Plastique: Manuel des maladies du tube digestif*. Debove, Archard et Castaigne. Vol. i, p. 707, Paris, 1907.
- ²⁰⁵ Salse: Hypertrophie de l'estomac et du côlon transverse. *Bull. Soc. anat.*, 1844, p. 79.
- ²⁰⁶ Sandifort: *Obs. Anat. Path.*, vol. iii, p. 11.
- ²⁰⁷ Sansoni, L.: Sul morbo di Brinton (Linite plastica, cirrosi dello-stomaco). *Torino*, 1894, Rosenberg et Sellier, 30 p. 2 pl. in. 8.
- ²⁰⁸ Schacher: Une observation du prétendue linite plastique. *Paris Thèse*, 1905, No. 297.
- ²⁰⁹ Schmidt: Atrophie généralisée. Cirrhose de l'estomac avec disparition des glandes à pepsine. *Revue médicale de l'Est*, 1881, 15 avril, et *Mémoires de la Soc. de méd. de Nancy*, 1881.
- ²¹⁰ Schnetter: *Deutsch. Archiv. f. klin. Med.*, Bd. xxxiv, S. 632.
- ²¹¹ Schoch: Über hypertrophische Stenose des Pylorus. *Inaug. Dis. Zurich*, 1857.
- ²¹² Schwartz: Linite probable, du petit cul-de-sac de l'estomac. *Bull. et Mém. Soc. de chir.*, 27 juillet, 1898, p. 814.
- ²¹³ Scott, N. S.: Non-malignant Stenosis of the Pylorus and Duodenum. *Cleveland J. M.*, 1897, ii, 1-12.
- ²¹⁴ Sheldon: *Cirrhosis of Stomach*. *ANN. OF SURG.*, vol. xxxix, 1904, p. 341.

- ²⁰⁴ Sheldon: A Further Report on a Case of Cirrhosis of Stomach. *ANN. OF SURG.*, 1906, vol. xlv, p. 666.
- ²⁰⁵ Siegheim: Über Syphilis des Magens. *Deutsch. med. Woch.*, vol. xxxvii, No. 4, Jan. 26, 1911.
- ²⁰⁶ Smith, J. W. F.: Cirrhosis or Fibroid Degeneration of the Stomach. *Edinburgh Med. Jour.*, vol. xviii, Part I, p. 521.
- ²⁰⁷ Snellen: Sclérose de l'estomac, article de la *Lancette Neerlandaise*, reproduit par le *Canstatt Jahrbuch*, 1856, iii, S. 302 (in Thèse Garret).
- ²⁰⁸ Soupault, M.: *Traité des maladies des l'estomac*. Paris, 1906, pp. 491-497.
- ²⁰⁹ Stretton: Leather Bottle Stomach. *Lancet*, 1909, pp. 464-465.
- ²¹⁰ v. Sury, K.: Beiträge zur kenntnis der totalen, einfach entzündlichen Magenschumpfung u. der fibrösen Polyserosthes (Zuckerguss). *Arch. f. Verdauungsk.*, 1907, vol. xiii.
- ²¹¹ Teissier: Pseudo-cancers de l'estomac. *Lyon médical*, 18 avril, 1886, p. 525.
- ²¹² Terrier: Néoplasme inflammatoire de la petite courbure de l'estomac, adhérent au lobe gauche du foie et à la paroi abdominale antérieure, pris pour un carcinome. *Laparotomie exploratrice palliative*. Guérison définitive de la malade. *Soc. de chir.*, 16 mai, 1894, p. 424.
- ²¹³ Terrier et Hartmann: *Chirurgie de l'estomac*, 1889, Paris, G. Steinheil.
- ²¹⁴ Testi-Zaccaria: Sulla natura dello linite plastica. *Riv. Crit. de Clin. Med.*, 1905, p. 601.
- ²¹⁵ Tilger: Über die stenosirende Pylorushypertrophie. *Virchow's Archiv.*, 1893, Bd. cxxxii.
- ²¹⁶ Tourlet: *La Linite Plastique*. Thèse de Paris, 1902.
- ²¹⁷ Trousseau: Article (gastrite chronique) in: *cliniques*, édition de 1873, t. iii, p. 74.
- ²¹⁸ Tuffier: Rapport sur une observation de M. Demoulin sur les tumeurs inflammatoires du tube digestif. *Bull. et Mém. Soc. de chir.*, 1899, p. 833.
- ²¹⁹ Tuffier: *Traitement chirurgical des rétrécissements du pylore*. *Pressé médicale*, 9 février, 1898.
- ²²⁰ Turner, F.: Fibrous Contraction with Hour Glass Stricture of Stomach. *Trans. Path. Soc.*, London, 1887.
- ²²¹ Van Leersum-Rotgans, J.: Extirpatie der gehulemang. *Œsophago-duodenostomie*. *Endogastritis obliterans*. *Deut. med. Woch.* 1. B. D. 67.
- ²²² Vautrin: Présentation d'un néoplasme fibreux de l'estomac. *Soc. de méd. de Nancy*, séances des 26 déc., 1900, et 9 janvier, 1901.
- ²²³ Vautrin-Hoche: *Considerations sur linite plastique*. *La Presse Méd.*, 1903, p. 477.
- ²²⁴ Verhoeghe, D.: Note sur un cas de linite plastique. *Echo méd. du Nord*. Lille, 1898, ii, 449-454.

- ²²⁵ Viti, A.: Contribuzione allo studio anatomo-pathologico della cirrosi gastrica. Boll. della Soc. tra il culori di Sc méd., v, 1887, resumée in Revue des sc. méd. de Hayem. T. xxxi, p. 543.
- ²²⁶ Voigtel, F. G.: Handbuch der pathologischen Anatomie. Halle 1804-5.
- ²²⁷ Weil et Péhu: Les sténoses pyloriques chez le nouveau-né et le nourrisson. Revue générale in Gazette des Hôpitaux, 1901, Nos. 112 et 115.
- ²²⁸ Weil et Destot: Observation publiée à la Société des Sciences médicales de Lyon, janvier, 1890, reproduite dans la Thèse de Garret.
- ²²⁹ Welch, W.: Pepper's System of Medicine, vol. ii, pp. 611-15, Ed. 1885.
- ²⁴⁰ Welch, W.: Johns Hopkins Bull., 1893, vol. iv, p. 98.
- ²⁴¹ Wilks: Malignant Fibroid Diseases of the Stomach. Trans. Path. Soc. of London, 1861-62, vol. xiii, p. 83.
- ²⁴² Ziegler: Lehrbuch der speciellen patholog. Anatomie, Jena, 1890, S. 506.

EXTENSIVE REMOVALS OF INTESTINE.

REPORT OF A CASE OF RECOVERY AFTER RESECTION OF TEN FEET EIGHT
INCHES OF THE ILEUM.

BY J. DAWSON WHITALL, M.D.,
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DA COSTA says ("Modern Surgery," 801) that the removal of more than six and two-thirds feet of gut will produce nutritional disturbances in an adult; that a child tolerates relatively larger amounts resected than an adult. Dressman reported 26 cases in which more than 3 feet 3 inches were removed, while Alexander Blaney (*Brit. Med. Journal*, Nov. 16, 1901) adds seven cases from literature and says that in nine of the 33 cases death occurred soon after operation. The late Nicholas Senn claimed that a third of the intestine was the extreme limit to be excised without inanition following. Roswell Park (*Buffalo Med. Journal*, Apr., 1903) reports the following resections in which over 6 feet 6 inches of intestine were removed, to which I have added the case herein reported.

OPERATOR	AMOUNT REMOVED	RESULT
(1) Koeberle	6 ft. 10 in.	Recovered.
(2) Kocher	6 ft. 11 in.	Recovered.
(3) Dressman	7 ft. 2 in.	Recovered.
(4) Shepherd	7 ft. 9 in.	Recovered.
(5) Kukula	7 ft. 9 in.	Recovered.
(6) Harris	7 ft. 10 in.	Recovered.
(7) Hayes	8 ft. 4½ in.	Recovered.
(8) Peck	8 ft. 5½ in.	Recovered.
(9) Lawers	8 ft. 9 in.	Recovered.
(10) Roswell Park	8 ft. 9 in.	Recovered.
(11) Payr	9 ft. 0½ in.	Recovered.
(12) Maydl	9 ft. 4 in.	Died three weeks later.
(13) Fantino	10 ft. 4 in.	Recovery.
(14) Monprofit	10 ft. 4 in.	Recovery.
(15) J. D. Whittall	10 ft. 8 in.	Recovery.
(16) Ruggi	11 ft. 0 in.	Recovery.
(17) Von Eiselsberg	11 ft. 8 in.	Died 25th day.
(18) Obalinski	12 ft. 2 in.	Died.

Several elements enter into the prognosis of these cases:

1. *The amount of gut left after the resection is variable*, as the entire bowel may vary from 15 feet 6 inches to 31 feet 10 inches (Treves). The length of the jejunum is about 8 feet or two-fifths of the small bowel; the ileum about 12 feet or three-fifths of the small intestine; the colon about 5 feet.

2. *The location of the portion removed*; the nearer the resection is done to the stomach the more serious the outcome, the most dangerous in order being duodenum, jejunum, ileum and lastly colon.

3. *Whether there has been fecal contamination of the peritoneum*; cases of course that have a rupture of the bowel and fecal extravasation have less chance of recovery because of the resulting septic peritonitis.

4. *Age and physical condition of the patient* has a great deal to do with the convalescence; the old bearing resection badly, while the reverse is true in the young. Patients who are suffering from malignant disease (sarcoma or carcinoma), who are tuberculous, who are generally "run down" have less chance of recovery.

AUTHOR'S CASE.—M. G., female, married, white, aged twenty-three years. About one year ago was confined, labor normal, but had a portion of the placenta retained, which later caused a pronounced sepsis. After a rather stormy convalescence (following a curettement) she finally recovered, but always complained of pain at the brim of the pelvis on the left side. This pain was often agonizing, causing her to faint several times. She last saw her menses four months ago, and considered herself normally pregnant. Symptoms of a detached placenta intervened, but she ran no temperature. On July 10, 1911, she had a convulsion (had had albumin and casts in the urine for some time) and was admitted to the Northwestern General Hospital. She was hurriedly prepared for a curettement by another physician. The placenta was immediately over the os uteri and came away easily, while portions of a macerated foetus (four months) were gradually removed. Then a portion of tissue was grasped and pulled down, when it was seen to be bowel.

Up to this time I had not seen the patient; she had been under ether two hours, but was in fairly good condition. The abdomen was hastily scrubbed up. A four inch incision was made in the median line and the uterus exposed; the gut was seen protruding from a perforation on the left anterior portion of the fundus uteri. The bowel was gently withdrawn from the uterus; it was then seen that the mesentery supplying that portion of the gut had been entirely stripped of its connection with the bowel. There was little if any bleeding from the mesentery (the vessels having retracted and clotted). The bowel was not torn or opened at any point. All the intestine that had no mesentery was resected; both ends were ligated, stumps touched with carbolic acid then alcohol; the ends invaginated with a purse-string suture. A lateral entero-enterostomy was then done. The torn mesentery was then whipped over and over with a continuous No. 3 chromic gut suture for its entire length. The distal end was about five inches from the cæcum. The hole in the uterus was then closed with a mattress suture. The abdomen was closed in layers.

The belly was filled with a quart of hot saline solution, while drainage consisting of a rubber tube was placed in the lower angle of the wound to the anastomosis; four pieces of plain gauze (two to the cul-de-sac and one to each side). Sterile dressings were applied. Time of operation was $1\frac{1}{2}$ hours, making a total of $3\frac{1}{2}$ hours under ether. During this time she was given $\frac{1}{30}$ gr. strychnine sulphate twice, and was very slightly shocked. Within fifteen minutes after she was put to bed she had reacted and was out of ether.

The specimen of gut removed was then measured in the presence of the physicians present and was found to be 10 ft. 8 inches.

The patient was placed on saline solution, after the method of Murphy, and given morphine sulphate gr. $\frac{1}{4}$. She had surprisingly little discomfort during her entire convalescence. The drainage was profuse and the wound had to be dressed daily. The Murphy treatment was discontinued after 48 hours. On the fourth day a triplex enema was given with very good result. For several days after, patient moved the bowels normally and passed a large amount of gas. Medication consisted of strychnine sulphate, gr. $\frac{1}{30}$ t.i.d. Nourishment was given every two hours—albumin water and broths for the first week, then soft diet,

and, after the nineteenth day, general diet. The drainage tube was gradually removed each day and a portion cut off, as was the gauze packing. All drainage was out by the eighteenth day, the wound cavity containing nothing but serum. The other portion of the incision healed by first intention. Patient left the hospital on the twenty-sixth day. Her bowels appeared normal in every respect. After being home for two days, as a result of her moving around too freely, she developed a phlebitis of the right femoral vein. Under rest, elevation, and the use of an ichthyol dressing (50 per cent.) the condition cleared up in ten days.

Granting that the ileum is about 12 feet, the patient should have about 1 foot 6 inches left of this portion of the small bowel. The writer realizes that even yet nutritional disturbances may follow, but up to the present time her physical condition is entirely satisfactory.

This case is reported with the desire that other seemingly hopeless cases of intestinal injury (or new growth) may have the benefit of extensive resection.

RIGHT INGUINAL HERNIA FOLLOWING APPENDECTOMY.

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FOR some time past the importance of preserving the nerves of the abdominal wall in doing laparotomies has been more or less realized, but it is doubtful if many know what the result of the destruction of one or more of these nerves means. This general subject has been gone over before in a previous paper published by the author in the *ANNALS OF SURGERY* for August, 1911. It was while working on that subject that the frequency of right inguinal hernia, after the apparent injury to one of the abdominal nerves in doing an appendectomy, became apparent. In order to establish more exactly the frequency of this condition, the records of St. Luke's Hospital, of New York City, were gone through and the history and physical examination of the last 190 cases of right inguinal hernia were looked through, in order to find out whether the patient had had an antecedent appendectomy. All congenital and double acquired hernias were naturally omitted.

For the privilege of using these records and reporting these cases, the author is indebted to Drs. C. L. Gibson and Robert Abbe, on whose services these patients were admitted to the hospital.

Altogether eight cases of right inguinal hernia were found in which there had been a history of an antecedent appendectomy, the time of appearance of the hernia varying, the shortest one being two weeks after leaving the hospital and the longest, four years. The greater proportion occurred in those cases in which the appendix wound had been drained, and in which, of necessity, it was larger, with a greater chance of

nerve injury, whether by directly cutting the nerve in doing the laparotomy or by the pressure on the latter by a drainage tube.

One case of special interest may be mentioned here, that of a young man, who was operated upon for acute appendicitis, the wound being drained. He stayed in the hospital six weeks, and was readmitted six years afterward with the history that his wound had given him no trouble until two weeks before admission, when it became swollen and tender. The old wound was then incised on two separate occasions, and considerable pus found between the layers of the abdominal wall. This healed up in about four weeks, and he left the hospital only to be readmitted in 15 days with a right inguinal hernia which he said he had noticed for the first time two or three days after his last leaving the hospital.

The reason for the appearance of these hernias is quite obvious. Practically all of the muscles in the region of the inguinal canal receive their nerve supply from the first lumbar nerve through its iliohypogastric and ilio-inguinal branches, the former supplying considerably more muscle, especially the lower part of the internal oblique and transversalis, commonly known as the conjoined tendon. The ilio-inguinal nerve is practically out of reach of the surgeon, emerging as it does just below Poupart's ligament about one and a half inches internal to and below the anterior superior spine of the ilium. The iliohypogastric branch, however, is generally more than one inch above the preceding, and runs downward and inward between the internal oblique and transversalis muscles. It crosses a line drawn from the umbilicus to the anterior superior spine almost two inches above the latter. Although the ordinary McBurney incision would run roughly parallel to the nerve, it can easily be seen in what danger the latter is, even though the fibres of the internal oblique are separated and not cut. The twelfth dorsal nerve takes the same course more than one inch higher up and is generally double.

Of course many cases are seen in which there is not only

a right inguinal hernia but also a hernia in the appendix scar. An example is given in one of the cases cited below. This naturally ensues from the cutting of the nerve, so that the muscle on the inner part of the wound being paralyzed is so flaccid that it not only admits the passage of a ventral hernia, but also causes a certain amount of retraction of the internal oblique and transversalis from Poupart's ligament, so that an inguinal hernia ensues.

The more essential points in the histories of the eight cases found in the hospital records are given below.

CASE I.—Appendectomy through McBurney incision for acute appendicitis with spreading peritonitis. Wound drained, patient left hospital in five weeks. Readmitted ten months from date of first operation with a hernia in the appendix scar, and a right inguinal hernia, the latter having first been noticed one month before. Left inguinal region normal.

CASE II.—Appendectomy through McBurney incision for acute appendicitis with general peritonitis, counterincision in loin. Both wounds drained. Patient left hospital cured in six weeks. Readmitted three years and two months from date of first operation with a right inguinal hernia, which had appeared thirteen months previously. Left inguinal region normal.

CASE III.—Appendectomy through McBurney incision for chronic appendicitis. Wound healed *per primam*, patient left the hospital in ten days. Readmitted in one month with a right inguinal hernia which had first appeared two weeks before. Left side normal.

CASE IV.—Appendectomy through McBurney incision for acute appendicitis. Wound drained, patient left hospital in four weeks. Readmitted in one month with a right inguinal hernia first noticed two weeks before. Left side normal.

CASE V.—Appendectomy through McBurney incision for chronic appendicitis, in hospital ten days. Readmitted four years from date of first operation with a right inguinal hernia which had existed one year. Left side normal.

CASE VI.—Appendectomy for acute appendicitis. Wound drained, patient left hospital in six weeks. Readmitted in four years, with a right inguinal hernia that had existed six months. Left side normal.

CASE VII.—Appendectomy for chronic appendicitis, McBurney incision. Left hospital in ten days. Readmitted in eighteen months with a right inguinal hernia that had existed two months. Left side normal.

CASE VIII.—The case of abscess in old appendix scar mentioned above.

Several other cases of this kind have lately been observed. One was a right direct hernia which had appeared one month after an appendectomy. This man had never had any trouble in the inguinal region before, and when seen the left side was normal. Another case was that of a young man who had had an appendectomy through a Kammerer incision, and noticed a swelling in the right groin three months after the operation.

Of course, eight cases out of 190 is not a large percentage, but the claim can justly be made that these are very conservative figures. It is very possible that there have been others out of the 190 who had trouble and who did not come back to St. Luke's Hospital to be operated upon for the hernia, or the fact of the antecedent appendectomy might have been overlooked by the historian. Yet, to the author's mind, this is a condition that really exists and, although recognized, enough attention has not been paid to it.

FRACTURE-DISLOCATION OF THE ATLAS.

BY F. L. CARSON, M.D.,

OF SHAWNEE, OKLA.

REPORTS of cases recovering after fracture-dislocations of the atlas are not infrequent, and my excuse in reporting this case is because recovery followed what was apparently a secondary myelitis, from which recovery seemed impossible, with the patient in such a desperate condition that operation seemed out of the question, and palliative measures alone were used.

CASE I.—W. T., aged twenty-nine, while riding on a roller coaster, standing erect, was thrown forcibly while at about the height of twelve feet, striking on his forehead, producing extreme extension. He was immediately stunned but not unconscious, was picked up, and with a little assistance went home. He complained of severe pain and stiffness of the neck, associated with swelling in the region below the right mastoid.

The patient was seen by various physicians before a correct diagnosis was made, after which a plaster jacket was applied, with relief from the pain and stiffness, from which he had suffered since the accident.

About two months after the accident the patient resumed his occupation as a street car motorman. He continued in this capacity without much inconvenience, until one cold, stormy night his car struck a drift, giving him a severe jolt and causing a return of the pain. His physician again applied the plaster jacket but this time without relief, and finally he was brought home, at which time I saw him in consultation, about four months after the injury.

Examination.—Well nourished male. His head turned to the left. Involuntary contractures of all four limbs. Breathing entirely diaphragmatic. Complete analgesia below the clavicle. Incontinence of feces and retention of urine. Hands and feet flexed. Marked prominence of posterior part of neck just below the mastoid. Bulging in the pharynx.

Patient was placed on his back and a collar around the chin and occiput applied, from which an 8-pound weight, over a pulley, was suspended. The head of the bed was raised about four inches, to overcome the traction. The pain at once became less severe, the paralysis after several weeks began to recede, first improving in the feet, then the legs, then the body, later the arms, and last of all the forearms. After allowing the patient to get out of bed an ambulatory extension apparatus was applied and worn for several months.

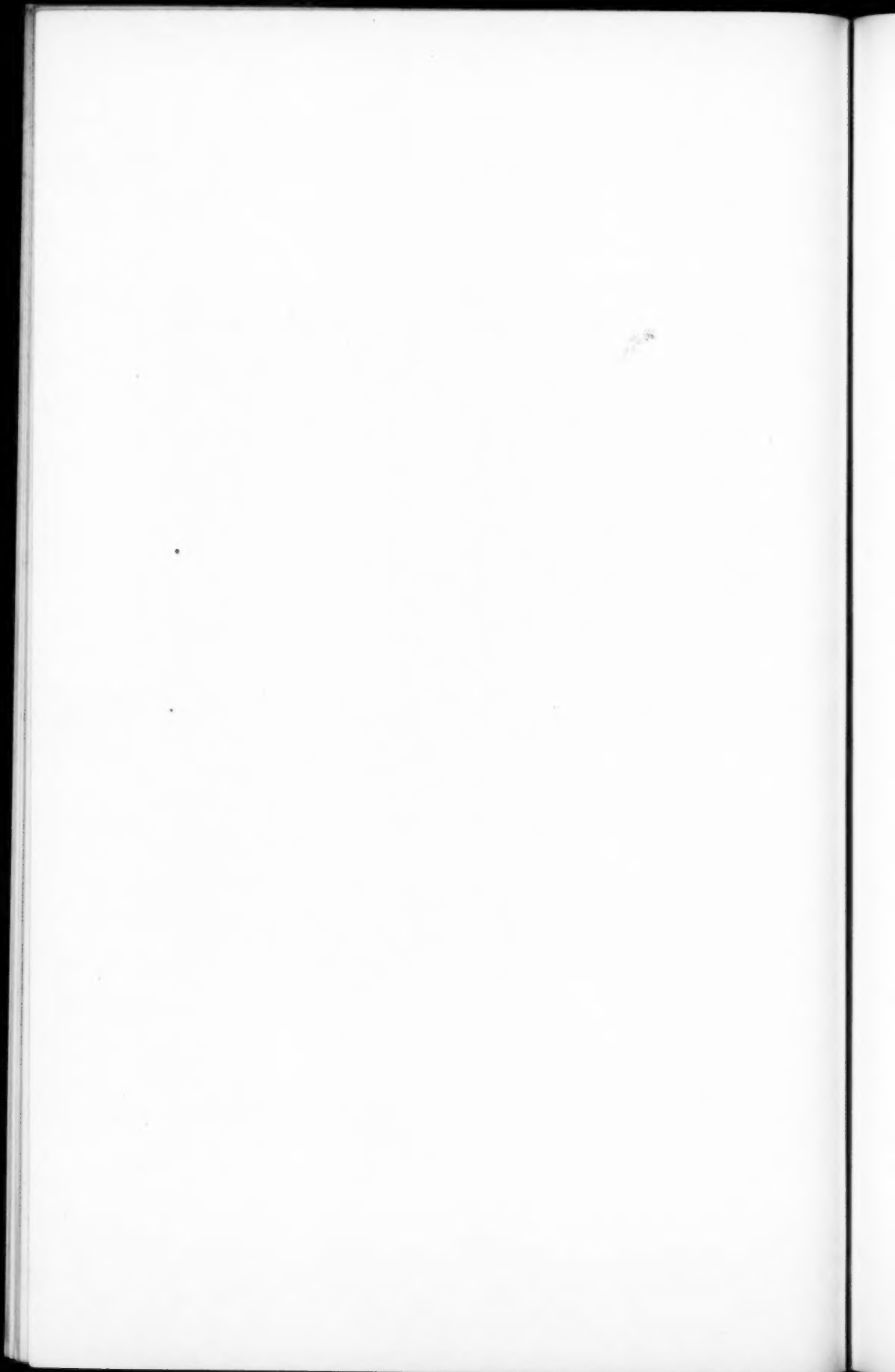
At the present time after nearly two years since the injury, the hands present evidence of paralysis. The fingers are clumsy, the interossei are atrophied, and the strength of the hands is very much impaired. The neck is much less rigid, there is no pain on attempts at motion. Swallowing food, at first difficult, is now easy. The patient at the present time is doing some manual labor, and is in fine general health.

The accompanying skiagram, while rather poor, shows the displacement to be marked. The recovery in this case, after a secondary myelitis, is unusual, and I believed worth reporting.

FIG. 1.



Fracture-dislocation of the atlas.



MUSCLE GRAFTING FOR GUNSHOT WOUND OF THE SHOULDER.

BY BENJAMIN BRABSON CATES, M.D.,

OF KNOXVILLE, TENN.

IF the end results of traumatic surgery are not always brilliant, the surgeon may console himself with the thought that, even if the cosmetic effect may not be all that is desired, the functional utility of the limb is restored oftentimes to a surprising degree even in apparently hopeless cases, so that in the face of discouraging prospects it is often well worth while to adopt an expectant plan of treatment. Because in so doing, as time goes on, some plan may suggest itself whereby other tissues may be substituted for the destroyed parts, and the surgeon not only may have the satisfaction of seeing his patient go out with a useful limb, but he may also feel repaid for the time and labor devoted to the individual whose limb he saved. Besides he is spared that compunction of conscience and the element of doubt as to the course pursued had he sacrificed an essential part of the body before trying conservative methods.

Such a case illustrating the point under consideration was brought to me by Dr. J. D. Norton of Wildwood Springs, Tenn., and which I hereby report as follows:

December 27, 1910, Leonard D., farmer, aged twenty-one, a fine specimen of physical manhood, was out hunting with an old fashioned breech loading hammer gun. Attempting to cross a picket wired fence, he thrust the barrel of the gun, muzzle first between the pickets, the butt of the gun resting on the near side of the fence. He then climbed over the fence, and as he did so, being a heavy man, he bent the fence forward. As he attempted to pick up the gun by the muzzle, the stock still being in the fence, the fence flew back and in so doing caught one of the hammers in the wire. As a result the gun was discharged; the load tore away the front of the man's axilla, the insertion of the pectoralis major, and the upper third of the biceps muscle;

it also made an oblique fracture of the surgical neck of the humerus.

The patient lived twenty miles in the country, consequently I did not see him until the next morning, as it was impossible to get him to the hospital earlier.

Examination showed a fearfully lacerated and contused wound, laying open the entire axillary space, exposing the brachial plexus of nerves, the main axillary vessels, and the deltoid muscle which were intact.

I nailed the fractured ends of the bone together (the nail subsequently loosened up and was removed) and packed the wound with corrosive sublimate gauze. The wound was kept open and packed with corrosive sublimate compresses in order to allow the devitalized tissue to slough and in order to watch developments, such as secondary hemorrhage, etc.

January 3, 1911, he had a slight hemorrhage from the anterior circumflex artery, which was controlled by compression. January 10, 1911, he nearly bled to death from sloughing of a branch artery near the brachial, as the blood ran down under him and was not noticed by the attendant. This was also controlled by packing. For twenty-four hours he hovered between life and death but finally rallied satisfactorily.

After all necrotic tissue had been thrown off and the supuration had nearly ceased and the wound had commenced to cicatrize, the problem became important to devise some way to give him a useful arm and to cover over the axillary space.

I did not wait for complete cicatrization before operating on account of the danger of scar tissue causing ischaemic paralysis and the probability of great difficulty in identifying the different structures.

In order to understand the steps taken to restore the function of the arm and shoulder, it will be necessary to remember the condition of things confronting us.

Here was a patient with the axillary space laid open, the axillary vessels and nerves exposed, the biceps nearly to the middle of the arm gone (making flexion of the forearm impossible), the head of the humerus ankylosed, and a fracture of its surgical neck.

It occurred to me that by resecting the head of the humerus with an inch or more of the shaft we could shorten the arm

FIG. 1.



Showing result of plastic to cover in defect of wall of axilla caused by a gunshot wound. I, ribbon of skin taken from arm; II, flap from chest. Note the degree of flexion of the forearm.

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enough to use, without tension, the coracobrachialis and anterior half of the deltoid muscles as fixed points of attachment for the biceps muscle if it were sutured to its upper end.

Having offered this plan of operation to the patient, he readily accepted the suggestion. So, February 8, 1911, I removed the ankylosed and carious head of the humerus and resected an inch or more of the shaft; then loosened the insertion of the coracobrachialis and the anterior half of the deltoid and sutured them with several strands of catgut to the freshened upper end of the biceps muscle. In order to lessen tension on the sutures the arm was kept in hyperflexion.

These different steps of the operation being completed, there still remained to cover over the axillary space. To do this a flap of skin was dissected from the front of the chest and slid over the axillary space and united with a narrow ribbon of skin crowded from the arm. The flaps readily united at their apposing edges and the raw surfaces exposed by their removal were allowed to granulate and cicatrize.

That the methods pursued to give this man a useful limb, and that the period of operation was well timed have been justified by the subsequent course of events.

The patient's arm is filling out and he says for carrying purposes is as strong as his sound arm. Of course the latitude of motion is not as great as before the injury and probably will never be; while he cannot carry his hand to the top of his head, he can, however, feed himself, wipe his face, and carry his fingers to the opposite shoulder with the injured limb.

The nerve supply of the arm and forearm is intact, and the radial artery at the wrist is unimpaired. In fact the upper limb from the elbow down is normal.

It is well in this connection to mention that at times he has associated movements—when he attempts certain motions, as in raising the shoulder, when flexing the forearm, and *vice versa*. In fact, Dr. Wm. R. Cochrane, who examined him at my request, could cause contraction of the fused deltoid and biceps by placing one pole of a galvanic current over the grafted deltoid and the other pole over the brachial plexus at the root of the neck.

The accompanying photograph (Fig. 1) taken July 8, 1911, shows the condition of the patient nearly seven months after the injury.

ON IMPACTED FRACTURES THROUGH AND NEAR THE FEMORAL NECK.

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THE main object of this paper is to call attention to the comparative frequency of impacted fracture near and through the neck of the femur, and to point to some features which may cause difficulty in diagnosis. The subject of injuries around the hip is time-worn, but many facts demonstrated by the Röntgen rays have not yet obtained due prominence in the current text-books on general surgery. Till comparatively recent years, the question of fracture near the hip resolved itself into distinguishing between "intracapsular" and "extracapsular" fractures of the femoral neck. Hard and fast distinctions were laid down whereby the one or other could be diagnosed, and outside the conventional types of injury diagnosis was doubtful. Intracapsular fracture of the neck of the femur was said seldom to be due to direct violence and rarely to be impacted. This teaching must have been responsible for many mistakes in diagnosis.

We are concerned here only with those injuries in which impaction occurs. These are almost always due to direct violence. In the event of direct violence being applied over the great trochanter, as in the common accident of a fall upon the hip, the force is transmitted along the femoral neck. This oblique force can be resolved into a horizontal crushing component which leads to impaction, and a vertical component which is most likely responsible for the upward displacement of the trochanter and shaft. According to the exact direction of the applied force, these horizontal and vertical components vary, and there result different kinds of fractures and displacements. In the case of indirect

violence, it is rare to get impaction primarily, though this may result from the fall upon the trochanter which frequently follows.

It is a law of mechanics that when a force acts transversely to a straight rigid rod fixed at one point, the breaking strain at any particular place is greater the nearer to that fixed point. In the case of a jar transmitted upward through the femur, the line of force passes vertically up through the top of the shaft and acts more or less transversely to the neck. The centre of rotation in the head may be taken as the fixed point, and the breaking strain along the neck will increase as the head is approached. This accounts for the fact that indirect violence usually causes a fracture of the neck just below the head (*subcapitalis*) and also for the occasional separation of the epiphysis of the head from a similar injury. It will be noted, however, that such indirect force only tends to produce vertical displacement, and there is no horizontal force which would lead to impaction.

The form of direct injury which causes an impacted fracture is usually a fall, whereby the hip strikes the ground, more rarely some heavy object falling on the hip may be responsible. The precise lesion resulting depends upon the age of the patient and the direction of the injuring force. Up to the age of twenty, at which time the epiphysis of the head should be joined to the neck, it is not unusual for the epiphyseal attachment to give way and a traumatic coxa vara to result, though fracture of the neck at this age is by no means unknown. During middle age and later years an impacted fracture of the neck or trochanter region results.

The position of the fractures cannot be foretold by considering the amount or kind of violence. Similar injuries may lead to fractures at different places.

For descriptive purposes we may divide impacted fractures in this region into: (1) impacted fractures through the neck of the femur wholly within the capsule; (2) impacted fractures through the base of the femoral neck or in the trochanter region. It must be understood that by an im-

pacted fracture we mean one in which the fragments are not movable on one another immediately after the accident. Many such a fracture becomes unimpacted owing to ill-directed manipulation. As soon as it is evident that serious injury has occurred, no rough handling should be undertaken before the X-rays have indicated the presence or otherwise of impacted fracture.

(1) Impacted fracture of the femoral neck within the capsule is not of such infrequent occurrence as the text-books would have us believe. A treatise on fractures published only last year states with regard to fracture of the neck of the femur: "rarely this form of fracture may arise from blows upon the trochanter." This statement perpetuates the traditional view held before the introduction of X-rays. It can be definitely asserted that it is comparatively common to find an impacted fracture of the femoral neck wholly within the capsule, resulting from violence directly applied over the great trochanter.

There are two groups of cases. The first conform more or less to the type description of "extracapsular" fractures, *i.e.*, shortening, eversion, and loss of power in the limb, but unless considerable violence is used crepitus will not be elicited. Bruising over the hip may or may not be a noticeable feature.

The second variety is atypical, and gives rise to varying deformity according to the direction of impaction. Slight shortening and various degrees of flexion, adduction, and inversion result. There are three reasons why an error of diagnosis may occur in this fracture. In the first place the shortening is but slight, half an inch or under being quite usual. Those who have done much measuring of lower limbs will appreciate the statement that differences of half an inch or under are not always easy to determine, for it may be difficult to locate the anterior superior spine accurately in a fat subject, and sometimes spasm of the muscles will prevent the limbs being adjusted symmetrically to the pelvis. Secondly, the impaction may produce an attitude of the limb not usually associated with fracture; for instance, in the

SKIAGRAM I.



Impacted fracture of neck of femur near the head, due to direct violence. (Case I.)

SKIAGRAM II.



Impacted fracture of neck of femur associated with adduction and flexion of thigh. (Case II.)

SKIAGRAM III.



Impacted fracture through base of the neck of the femur (Case III). Skiagram taken five days after the accident.

SKIAGRAM IV



Fracture through the base of neck with impaction but no shortening. Arrows point to lines of secondary fracture. See Fig. 1 and Case V.

SKIAGRAM V



Impacted fracture of base of neck. The common type of injury. The detached top of great trochanter can be seen displaced inward. See Fig. 2.

SKIAGRAM VI.



Impacted fracture through base of neck of femur. Third type of injury. See Case VII and Fig. 3.

second case quoted below, the position assumed by the limb resembled that due to a dorsal dislocation of the hip. Thirdly, the loss of function at the hip may not be complete. It may be possible for the patient to lift the limb off the bed or even to bear the weight on it and walk about, though limping.

As illustrations of the two groups of cases the following will serve:

CASE I.—W. P., aged sixty-five, was knocked down and fell on the right hip. On admission (September, 1910) there was eversion and slight abduction of the right lower limb, which was half an inch shorter than the opposite member. Pressure over the great trochanter was painful, but no bruising was visible. The iliotibial band was slacker than on the sound side. Voluntary movement at the hip-joint was just possible, but painful. Skiagraphy showed an impacted fracture of the femoral neck near the head, and wholly within the capsule (see Skiagram I).

This illustrates the first type. The second group is exemplified in the next case.

CASE II.—W. S., aged thirty-eight. Fell off a bicycle on the right hip three weeks before admission. For a short time he rested, but soon got about again, though he limped painfully. His injury was not treated as a fracture, and no improvement took place. On admission (August, 1910) he was still able to walk, though with difficulty. The right lower limb was three-eighths of an inch shorter than the left, and was kept in a position of flexion and slight adduction. Abduction was impossible. Rotation outward and inward was limited and painful. There was pain on pressure over the great trochanter, which was no nearer the middle line than that of the opposite side. A skiagram showed a fracture of the neck within the capsule and just below the head. The neck was impacted tightly into the upper part of the cancellous tissue of the head in such a way as to account for the adduction and flexion of the limb (see Skiagram II).

In both the above cases movement at the hip-joint was possible though attended by pain. In the second patient the position of the limb simulated dorsal dislocation, but by considering the nature of the injury, the small amount of shortening, and the rather atypical deformity, a diagnosis was possible apart from the X-ray finding.

Owing to the kindness of Dr. Harrison Orton I am able to include a skiagram of another case.

CASE III.—Patient fell off a bicycle on to the hip. He worked for two days afterwards, getting about his ordinary vocation though with some pain. Then he was compelled to take to bed. The skiagram was obtained five days after the accident. It shows a tightly impacted fracture through the neck (see Skiagram III).

It is quite possible that some cases of fracture through the neck caused by indirect violence (*i.e.*, the common fracture in very old people) may become tightly impacted because of an immediate subsequent fall upon the trochanter of the affected side. In most of the cases I have seen, though chiefly affecting elderly people, this causation was negatived by the nature of the accident. The following case, which I am able to give owing to the courtesy of Mr. Warren Low, illustrates this possibility. I can do no better than give the account in Mr. Low's own words.

CASE IV.—“The patient was an active old gentleman aged seventy-three, who, two years before I saw him, whilst travelling in Italy, tripped and fell upon his trochanter. He continued his sightseeing for the day, but stayed in bed for the next five days. He was afterwards able to get about, visiting Florence and Rome. He had been seen by an Italian doctor who assured him that nothing was broken. This assurance was repeated in Paris where he again sought advice, and again when he reached London. I may mention that he stated that, some time after the accident, while travelling to Paris in a jolting train he felt a sudden sharp pain in the hip region, and ever after that the hip gave him more trouble. I saw him two years after the accident, and

whatever may have been obscure before, was then quite obvious. The movements round the left hip-joint were quite free with the exception of internal rotation and abduction, but active movements though free were feeble. The limb was one and a half inches shorter than its fellow, and the trochanter was above Nélaton's line. Moreover, on rotating the femur, the trochanter rotated on its own axis and not through the arc of a circle of which the neck of the bone was the radius. It was not a difficult matter to diagnose a fracture of the neck of the bone with a false joint, and this was confirmed by skiagraphy."

This case must have been a tightly impacted fracture at the first, else it could not have been altogether missed by the doctors who saw him at or soon after the occurrence of the accident.

(2) Impacted fracture through the base of the femoral neck and the trochanter region includes all those which used to be loosely called "extracapsular fracture of the neck," a terminology anatomically incorrect and clinically of doubtful value.

From outward appearance it is often difficult or impossible to tell whether this fracture or an impacted fracture near the head has occurred. Both occur in middle or old age, and result from the same kind of violence. Perhaps slightly greater force is responsible for fracture through the base of the neck, for shortening and eversion of the limb and bruising over the trochanter are more marked in these cases. Owing to this greater bruising and more severe injury, movement at the hip is more uncommon. But the mechanism of the two fractures is quite different. In fracture through the neck the firm tissue of the lower part of the neck is driven and tightly impacted into the cancellous tissue of the upper part of the head; in fracture through the base of the neck the cancellous tissue of the great trochanter is split up by a firm wedge of bony tissue belonging to the cervix femoris. This wedging of the cervix into the trochanter region is responsible for two or three secondary fractures which frequently complicate the primary breakage (see Fig. 1).

One is due to vertical splitting of the great trochanter, and usually separates off the posterior part of the great trochanter and the adjoining bone, including the lesser trochanter and even a little of the shaft beyond; another is horizontal, and detaches the top of the great trochanter from the previous fragment. Occasionally, and probably from a greater degree of violence, the lesser trochanter and a flake of adjacent bone from the shaft are split off, owing to the cutting action of the lower compact tissue of the neck.

It has been pointed out by Ralph Thompson that the *calcar femorale* probably plays an important part in the causation of these secondary fractures. The *calcar* is a vertical plate of more compact bone standing upright in the midst of the cancellous tissues of the femoral neck. Its base is attached to the inner and under surface of the neck as far down as the lesser trochanter, and its outer edge points like a vertical razor edge towards the great trochanter. When a force is applied over the hip, this firm blade tends to split the great trochanter in a more or less vertical direction. From a study of skiagrams it would appear likely that the upper layer of compact tissue of the neck acts as a blade to produce the secondary horizontal fracture. It seems likely that the lower layer of compact tissue of the neck is responsible for the occasional splitting away of the lesser trochanter and adjacent portion of the shaft.

Three grades of injury have come under my notice. In the first a primary fracture occurs at the junction of the head and neck, and the base of the neck is then thrust into the cancellous tissue of the great trochanter. The lines of the secondary fractures (horizontal and obliquely vertical) can be seen, though no separation of the fragments takes place (see Fig. 1 and Skiagram IV).

The second grade shows a more advanced condition. The firm tissue of the neck causes separation of the fragments of the trochanter along the lines previously mentioned. The base of the neck loses its support, and marked shortening occurs. The tip of the great trochanter is nearly always

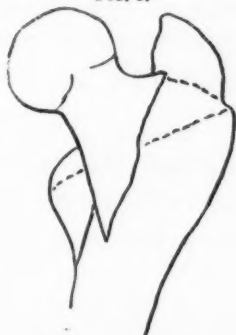
drawn inwards toward the upper rim of the acetabulum. This is probably due to the action of the numerous muscles attached to its upper border. This grade of injury is the most common type of fracture resulting from a fall on the hip (see Fig. 2 and Skiagram V).

A third type of injury is shown in Fig. 3 and Skiagram VI. In it the process is carried one stage further, for the small trochanter and a slice of shaft are also split off.

The following are illustrative of the three types:

CASE V.—J. B., aged forty-two (admitted February, 1911). Gave history of stumbling and falling heavily upon the left hip

FIG. 1.



Showing lines of primary and secondary fracture in Case V.

FIG. 2.



Showing lines of separation in common type of fracture through base of neck.

as he was trying to get into a train. When admitted, there was eversion and slight adduction of the left lower limb. No shortening was obtained, but there was definite relaxation of the iliotibial band. Some indefinite crepitus was felt on moving the femur, but this was elicited only once or twice. The patient could move his limb at the hip, though with some pain. The skiagram showed a fracture through the base of the neck which ended below in the lesser trochanter. The dense neck had been firmly impacted into the cancellous tissue of the upper portion of the shaft, and had begun to split off the upper part of the great trochanter. No shortening was obtained, since there was no alteration of the angle of the neck with the shaft, and no displacement in a vertical direction. The only positive

sign was relaxation of the iliotibial band (see Fig. 1 and Skiagram IV).

The effects of still greater violence, or of similar violence acting on an atrophic bone, are shown by the following:

CASE VI.—E. S., aged seventy-nine. Was accidentally knocked down by a blind man and fell upon her left hip. On admission, pain made examination difficult. The left lower limb was everted, and there were two inches of shortening. Voluntary movement was just possible, but very painful. The X-ray negative revealed an impacted comminuted fracture through the trochanters. The neck had evidently been wedged in between the two trochanters and separated portions of both. The appearance was exactly as if a further amount of violence had been applied to the previous case. The angle between the neck and shaft had been diminished almost to a right angle. See Skiagram V and Fig. 2.

This case shows why shortening varies so greatly in fracture through the base of the neck. When the force is only of moderate severity, the neck is firmly fixed by its impaction into the neighboring cancellous tissue; but when the impact is severe enough to break up the trochanter, resistance to vertical displacement disappears, and the vertical component of the injuring force leads to considerable displacement and shortening.

In view of the fact that the top of the trochanter is drawn inward, it might be advisable to put some of these fractures up in an abducted position. Skiagram V and Fig. 2 show a similar condition, though not taken from this actual case.

A still further degree of violence occurred in a case more recently in St. Mary's Hospital.

CASE VII.—Patient fell a distance of 25 feet upon the right hip. On admission there was much bruising over the right hip, and one and a half inches of shortening. The iliotibial band of fascia was relaxed on the affected side. Crepitus was elicited.

The limb lay in an everted position and no voluntary movement was possible. There was a swelling in the upper part of the thigh. A skiagram showed a fracture through the base of the

FIG. 3.



Showing the third type of fracture through base of neck (Case VII).

neck with separation of the lesser trochanter and a slice of the adjacent shaft (see Fig. 3 and Skiagram VI). The lower end of the cervical wedge caused this detachment.

CONCLUSIONS.

1. Impacted fracture of the neck of the femur (within the capsule) is by no means an uncommon occurrence.
2. It results from direct violence applied over the trochanter major, and may permit of considerable voluntary and passive movement of the hip without crepitus.
3. An unimpacted fracture resulting from indirect violence may occasionally be converted into an impacted fracture owing to an immediately subsequent fall upon the affected hip.
4. The impaction may cause an atypical deformity, even sometimes simulating dorsal dislocation.
5. Fracture of the base of the neck is always primarily impacted; if the force applied is great, there are always secondary fractures, both vertical and horizontal, through the trochanter region.

6. Persons with impacted fractures of the femoral neck or base of the neck may and often do walk about for some days after the accident.

7. An impacted fracture of the base of the femoral neck may exist without appreciable shortening when the force is not sufficient to cause the trochanter region to be split up by the wedge-like cervix femoris.

In conclusion I wish to express my indebtedness to Dr. Harrison Orton for his kindness in allowing me to make use of all the radiographs inserted.

NOTE ON CATGUT STERILIZATION.

A PRELIMINARY REPORT.

BY WILLARD H. HUTCHINGS, M.D.,

OF DETROIT.

SEVERAL years ago the writer began an experimental study of the various methods which have been employed for the sterilization of catgut. The longer I worked with them the more I became convinced that the secret of catgut sterilization lay in the complete removal of all traces of water. After trying a great many methods, most of which failed, the following was evolved which will sterilize catgut without impairing its physical qualities. The point of the method lies in the way the water is removed. This is done in a vacuum over sulphuric acid. It is a well-known physical fact that in a vacuum of less than 4 mm. of mercury water vaporizes. For example, a piece of ice placed in a desiccator under less than 4 mm. pressure will remain ice until it completely disappears without liquefying. The sulphuric acid absorbs the water vapor as fast as it is formed. It is also well known that in the absence of water very few, if any, chemical changes take place. Therefore a perfectly dry catgut can be heated nearly to the charring point without materially injuring it. It has also been proved by repeated experiments that 150° C. dry heat for two hours will kill all germs, with few exceptions, these being non-pathogenic. This is the, almost universal, method employed by bacteriological laboratories to sterilize infected glassware, etc.

Based on these principles, the method I employed is as follows: Raw catgut without any previous treatment is placed in suitable containers which are left open. In my work I have employed a simple straight glass tube sealed at one end, the other being open. These tubes are then placed in an ordinary vacuum desiccator, the bottom of which is covered with sul-

phuric acid and connected with a vacuum pump. The air is then exhausted until a pressure of less than 4 mm. is secured, the more perfect the vacuum the better the result. When this is attained the desiccator is closed, disconnected from the pump, and set aside. Eight days are long enough to secure dryness. At the end of this time air is allowed to enter the desiccator. If perfectly dry air is needed it can first be passed through Wolff bottles containing sulphuric acid. The tubes are then rapidly sealed and are ready to sterilize. Any method of heating may be employed. Hot air ovens have been found uncertain, as it is very difficult to regulate the temperature. In my work I have employed a mixture of glycerin and distilled water with a boiling point of 150° C., the container being fitted with a reflex condenser. The sealed tubes are placed in this and boiled two hours, thus securing two hours of dry heat at 150° C.

Any method of sterilizing catgut must fulfil certain requirements. First it must render the catgut sterile. It is not sufficient that it kill the various micrococci, colon bacillus, typhoid bacillus, and possibly anthrax. It must absolutely destroy every germ which may in any remote way produce infection. And it must do it every time. Sterility is the prime requisite. In addition, the physical characteristics of the catgut should not be impaired. It must be strong, elastic, absorbable. In order to test the sterilization of gut prepared in this way, the following experiments were undertaken: Number 4 catgut was cut in one and one-half inch lengths and placed in 250 c.c. Erlenmeyer flasks, containing suitable culture media. These flasks were then inoculated with "hay" bacillus, "potato" bacillus, anthrax, tetanus and dirt from the street (aërobic and anaërobic) and incubated at 37.5° C. for 21 days. At the end of this time they were removed and submitted to the sterilizing process above described. One hundred and fifty samples of each kind of infected gut were used. They were then planted in suitable culture media and incubated for 14 days. Not one of the samples gave a growth. There were also no contaminations. In order to be perfectly sure that they

were sterile, twenty-five samples of anthrax and tetanus gut were transplanted into guinea pigs. In no instance did they produce anthrax or tetanus in the animal. Control animals which were inoculated with the gut before sterilization died of the diseases.

The tensile strength of the gut was tested in the following manner: Samples of gut one foot in length were attached at one end to a self-registering scale, at the other to a windlass in such a manner that the strain could be applied steadily. Force was applied until the gut broke. In this manner one hundred samples of gut were tested before treatment and one hundred after sterilization. The raw gut gave an average breaking strain of 8.7 pounds, while the sterilized gut gave an average of 8.9 pounds. In other words, the sterilized gut was a little stronger than the raw gut. This is in accordance with the well-known fact that a dry piece of wood, for example, has a higher tensile strength than a green piece.

Tests for absorbability were made on guinea pigs and showed that No. 2 gut was absorbed in nine days. The appearance and elasticity of the gut is not altered by the process of sterilization. Instead of being roughened during sterilization it is even smoother than before. The following conclusions are justifiable:

1. That gut prepared by this method is sterile.
2. That its tensile strength is not impaired.
3. That it remains in the tissues long enough to fulfil the purpose for which it is intended.

Unfortunately the method is not simple. It requires rather complicated apparatus and a first-class vacuum pump. The above is in the nature of a preliminary report. It is intended to experiment further in an effort to not only simplify the method but to secure wider applications if possible.

THE TREATMENT OF LACERATED AND INCISED WOUNDS OF THE EXTREMITIES. *

WITH A REPORT OF FIVE TYPICAL CASES.

BY JAMES A. KELLY, M.D.,
OF PHILADELPHIA.

Visiting Surgeon to St. Mary's Hospital; Associate in Surgery, and Pathologist to the Philadelphia Polyclinic Hospital and College for Graduates in Medicine.

ALTHOUGH marked advances have been made in the technic and treatment of abdominal conditions, our attention is often attracted to the fact that the treatment of lacerated, incised, and punctured wounds of the extremities has not kept pace with the work done in the other branches of surgery, and while the percentage of cases that were treated by primary amputation for destruction of the arterial or nerve supply, or by secondary amputation for gangrene due to thrombosis of infection, is not so great, the fact remains that the ultimate result, as shown in the loss of function, muscular atrophy, contractures, and the often-marked involvement of sutured tendons, nerves, and blood-vessels in one mass of cicatricial tissue, is very poor. This condition greatly limits the usefulness of the individual, decreases his earning capacity, and too frequently ends in prolonged lawsuits for indemnity. It is with the object of bringing this common condition before the attention of the Fellows that I wish to present this report of five typical cases.

CASE I.—*Cartridge shell wound of the arm, involving the brachial artery, basilic vein, and median nerve; circular arteriorrhaphy, circular phleborrhaphy, and neurorrhaphy.*

Mrs. A. G., sixty-six, w. housewife, Germany, admitted to St. Mary's Hospital July 4, 1910. Patient, while walking along the street July 4, 1910, heard an explosion as a trolley car passed and at the same time felt something strike her in the upper arm,

* Read before the Philadelphia Academy of Surgery, May 1, 1911.

accompanied by a sharp stabbing pain in the same region which radiated down the forearm to the hand and fingers. The patient noticed a small amount of bleeding from the wound, and on account of this and the pain went to the hospital.

On admission the arm was thoroughly cleansed and an antiseptic dressing applied. The wound was not considered to be of great importance by the resident physician and he did not notify me until the next morning, when examination showed complete paralysis of the muscles supplied by the median nerve and apparently no injury to other important structures. The arm showed a small transverse wound 1 to 1.5 cm. long on the anterior surface of the right arm about the junction of the middle and upper thirds, through which there was a small amount of blood oozing. The entire inner aspect of the arm was swollen over an area of about three to four inches and was ecchymotic. Radial and ulnar pulses were distinctly palpable, but not as full as on the left side. An X-ray examination showed the presence of a small foreign body about 8 to 10 mm. square.

Under ether anæsthesia longitudinal incision $5\frac{1}{2}$ inches in length was made, with its centre at the wound of entrance. On cutting through the deep fascia a large blood-clot was evacuated, and this was followed by a gush of arterial blood. A tourniquet above the wound and a careful dissection showed a transverse wound of the brachial artery involving half of its calibre, almost complete severance of the median nerve, and a transverse wound of the basilic vein involving its entire calibre. Further dissection revealed a small piece of a cartridge shell about 1 cm. square imbedded in the coracobrachialis muscle. The wound was thoroughly irrigated with hot normal salt solution, the edges of the wound of entrance excised, and the wounds in the brachial artery and basilic vein were closed by circular arteriorrhaphy and phleborrhaphy by Carrel's method. The cut ends of the median nerve were united by means of two fine silk sutures passed directly through the nerve. The operative wound was then closed with continuous catgut sutures uniting the deep fascia and interrupted silkworm gut through the skin. Drainage was provided for through a small stab wound about two inches above the internal condyle, using rubber dam. A dry sterile dressing and an internal angular splint were applied. The wound healed by primary union; the drain was removed in 48 hours, and the

sutures at the end of eight days. The patient was discharged at the end of three weeks and recommended to return for massage and passive motion.

Examination three months after operation showed normal pulsation of brachial and radial arteries, marked atrophy of flexor muscles, anæsthesia over areas supplied by median nerve; marked changes were present in the skin of the hand, particularly the fingers and thumb being thin, smooth, shiny, and cold; the nails were dry, dark in color, striated longitudinally, and there was marked sweating of the palm of the hand. There was marked stiffness of the elbow-joint in a position of semi-flexion, and also of the wrist and phalangeal joints. Voluntary flexion was absent in the fingers, was weak at the wrist-joint, and pronation of the hand was impossible. The patient complained of a general pain throughout the forearm and hand on attempts at movement.

Examination six months after operation showed a moderate return of sensation, muscular power, and a lessening of atrophic changes.

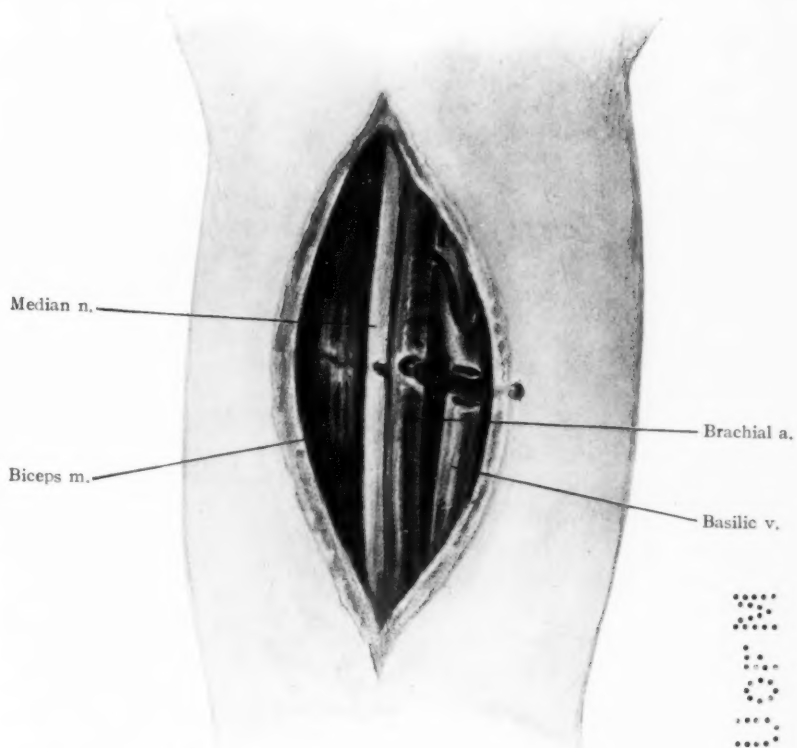
Examination March 30, 1911, showed a moderate degree of muscular atrophy, motions at elbow and wrist free, pronation and supination normal, flexion of fingers fair, still some trophic changes present over terminal phalanges, nails becoming smooth and normal in color at bases. No pain present on motion. General condition satisfactory.

CASE II.—*Lacerated wound of arm, severing biceps, portion of brachialis anticus, brachial artery, basilic vein, median and ulnar nerves; arteriorrhaphy, phleborrhaphy and neurorrhaphy.*

Jacob G., sixty-six years old, U. S., shuttle maker, admitted to St. Mary's Hospital September 23, 1910, at 2 P. M. Patient while at work had his clothing caught in a portion of the machinery and received a lacerated wound of the right arm from a circular saw. Admitted to the hospital in a profound state of shock.

Examination on Admission.—Patient in a profound state of shock. On the right arm there was a lacerated wound about six inches in length, extending from the junction of the middle and upper thirds on the external surface running downward and inward. Inspection of wound showed the biceps muscle, the brachialis anticus muscle, brachial artery, the basilic and cephalic veins, the median and ulnar nerves to be completely severed and

FIG. 1.



Lesion present after exposure by incision. (Case I.)

23

FIG. 2.



Shows healed wound with scar of punctured wound at centre. Hand in position of full extension. Note the degree of atrophy still present. (Case II.)

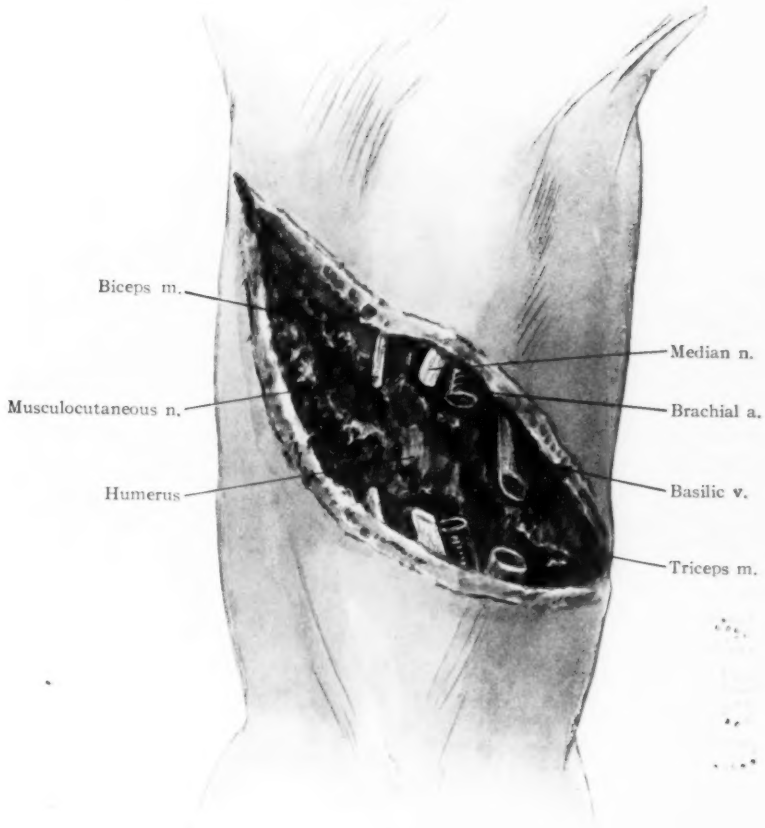
FIG. 3.



Shows the degree of flexion present, which is limited entirely by the stiffness present in the phalangeal joints. (Case I.)



FIG. 4.



Showing the extent of the lacerated wound. (Case II.)

30
10
0
100

the cut ends retracted, and the wound partially filled with blood-clot. The bleeding had been temporarily controlled by means of a cloth tourniquet. The patient was given an intravenous injection of one litre of normal salt solution, a shock enema and a temporary antiseptic dressing placed around the arm. At the end of three hours the patient had sufficiently reacted from shock to warrant operative intervention.

The patient was given morphine $\frac{1}{4}$ gr., atropine 1/100. The arm was thoroughly cleaned with soap and water and alcohol 70 per cent., and the wound irrigated with hot normal saline solution. Circular arteriorrhaphy of the brachial artery and circular phleborrhaphy of the basilic vein were performed, using the method of Carrel. It was then found necessary to give the patient a little ether to continue the operation. The cut ends of the median and ulnar nerves were sutured with fine silk by the direct method, and the cut portions of the biceps and brachialis anticus muscles were sutured with No. 1 interrupted chromic catgut, the fascia and skin being united with interrupted sutures of silkworm gut and drainage provided for at the lower angle of the wound with rubber dam. A sterile dressing and an internal angular splint were then applied. The patient was fully stimulated, and in spite of everything that could be done he failed to react and died about ten hours after leaving the operating room.

CASE III.—*Incised wound of the arm involving the brachialis anticus muscle; the tendon of the biceps, the basilic vein, the median and ulnar nerves, tenorrhaphy, myorrhaphy, neurorrhaphy.*

M. McD., sixteen years of age, schoolboy, admitted to the Polyclinic Hospital, September 8, 1910, service of Dr. Louis A. Steinbach, to whom I am indebted for the privilege of reporting this case.

Patient while painting the outside of the window frame was supporting his weight with his hand against the window pane, when the latter suddenly gave way and the patient partially fell through the window severely cutting his right arm with a piece of the broken glass. Admitted to the accident room of the hospital in a state of shock with a tourniquet around the upper part of the arm.

Examination on admission showed patient to be in a moderate degree of shock. The right arm presented an irregular in-

cised wound about four inches in length, beginning at the junction of the middle and lower thirds of the arm on the anterior surface running downward and inward. Retraction of the edges of the wound showed complete severance of the tendon of the biceps, a portion of the brachialis anticus muscle, the basilic vein, the median and ulnar nerves. Under ether anæsthesia the arm was cleaned with soap and water and alcohol 70 per cent., and the wound irrigated with hot normal saline solution. The separated portions of the brachialis anticus muscle and the tendon of the biceps muscles were sutured with No. 1 chromized catgut, the divided median and ulnar nerves were united with interrupted sutures of fine silk, the cephalic vein was ligated, and the fascia and skin closed with interrupted silkworm gut sutures. Drainage was provided for at the lower angle of the wound, and a dry sterile dressing applied. The wound showed a marked degree of infection several days after operation, which necessitated the removal of several of the sutures. The patient was discharged to the out-patient department for subsequent treatment September 24, 1910, sixteen days after admission. (Dr. Butler, Chief Resident.)

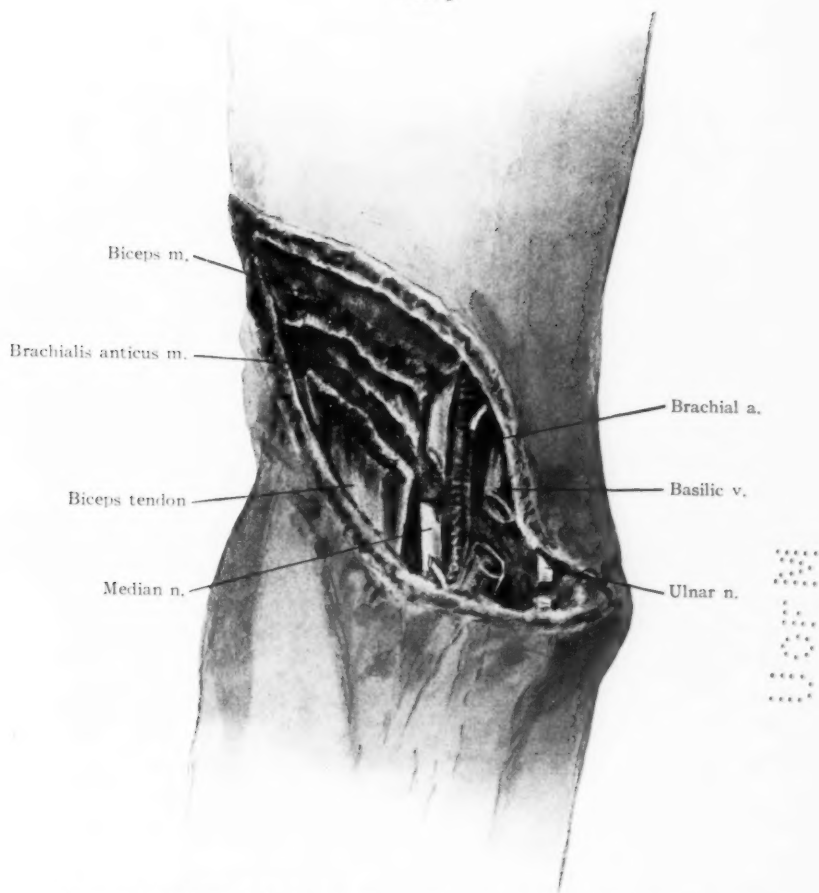
Examination March 30, 1911, showed normal flexion at the elbow, restricted motion of the wrist and of the fingers, most marked in the ring and middle fingers, still loss of sensation of index, middle, ring, and little fingers on flexor surfaces. Pronation and supination were still impaired.

CASE IV.—*Incised wound of forearm, flexor surface, involving the tendons of the flexor carpi ulnaris, palmaris longus, flexor sublimis digitorum, flexor profundus digitorum, excepting the division to the index and middle fingers, the ulnar artery, and the median ulnar nerves; multiple tenorrhaphy, neurorrhaphy.*

E. P., thirty-seven years of age, machinist. Accident occurred January 18, 1911. Patient, while at work at Cramp's shipyard, was struck on flexor surface of right wrist by a piece of a falling arclight globe.

Examination on admission showed a transverse incised wound about two inches in length on the flexor surface of the right forearm one inch about the hand. Through the separated edges of the wound were seen the cut ends of the ulnar and median nerves, the cut ends of the ulnar artery, the divided ends of the tendons of the flexor carpi ulnaris, the palmaris longus, the flexor

FIG. 5.



Showing the extent of the lacerated wound and the involved structures. (Case III.)

1000

FIG. 6.



Showing the healed wound, the muscular atrophy, and the degree of extension of the hand. (Case III.)

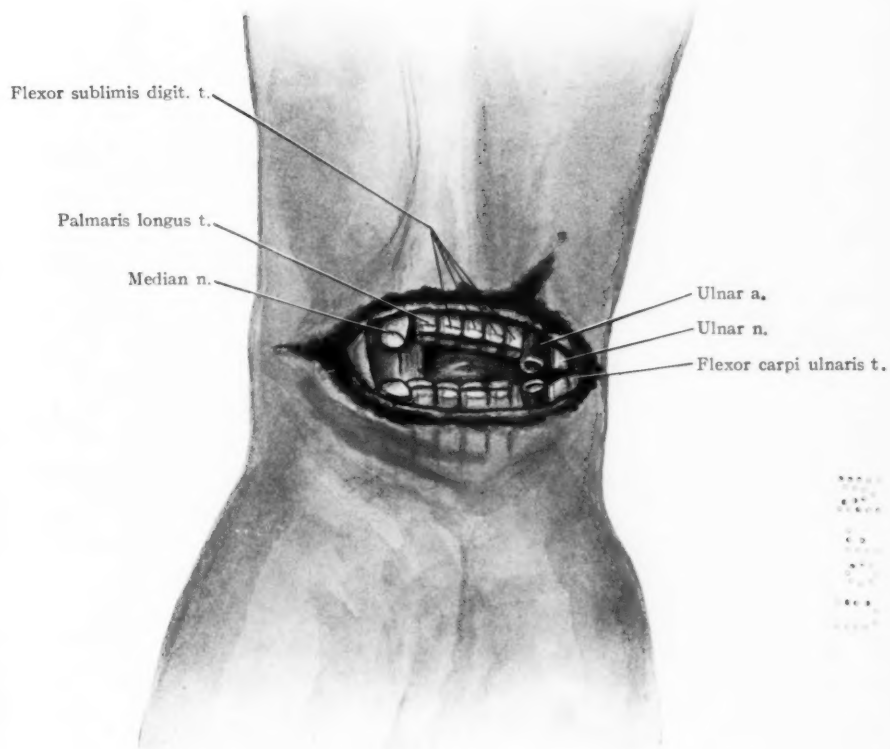
FIG. 7.



Showing the degree of flexion possible. (Case III.)

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 834 835 836 837
 838 839 840 841

FIG. 8.



Showing the extent of the incised wound and the involved structures. (Case IV.)

3840

FIG. 9.



Showing the healed wound and the amount of extension. (Case IV.)

FIG. 10.



Showing the degree of flexion possible. (Case IV.)

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1900

sublimis digitorum, and the flexor profundus digitorum, excepting the divisions to the index and middle fingers.

The operation was performed by the Resident Physician, Dr. McBride, without an anæsthetic, as the patient refused an anæsthetic. The arm was cleansed with soap and water and alcohol 70 per cent., and the wound irrigated with hot normal saline solution. The severed tendons were united individually with two interrupted sutures of silk, the cut ends of the nerves were united with interrupted silk sutures, the ulnar artery was ligated, the wound closed with interrupted silkworm gut sutures, a dry sterile dressing applied, and the hand dressed in extension on a splint. The wound healed by primary union, the sutures were removed on the eighth day and the splint at the end of two and a half weeks. Massage and passive motion were then instituted.

Examination March 30, 1911, showed the affected tendons to be firmly surrounded by scar tissue and united with the cicatrix of the skin; flexion of all the fingers was limited, particularly that of the ring and little fingers; cutaneous anæsthesia of hypotenar portion of the palm of the hand and the outer side of the ring and of the little fingers. There was marked tremor of the hand, which was cold and perspiring; the finger nails were moderately darkened, irregularly ridged, and longitudinally striated. The skin of the hand was thin, shiny, and bluish white. The scar was very tender and supersensitive. (In this case there is evidently separation of the cut ends of the ulnar nerve and a secondary nerve suture will be required.)

CASE V.—*Incised wound of the flexor surface of the forearm one inch above the wrist-joint, involving the tendons of the flexor carpi ulnaris, the palmaris longus, the flexor carpi radialis, the flexor longus pollicis, the flexor sublimis digitorum, the flexor profundus digitorum excepting the divisions to the ring and little fingers, the median and ulnar nerves, and the ulnar artery; multiple tenorrhaphy and neurorrhaphy.*

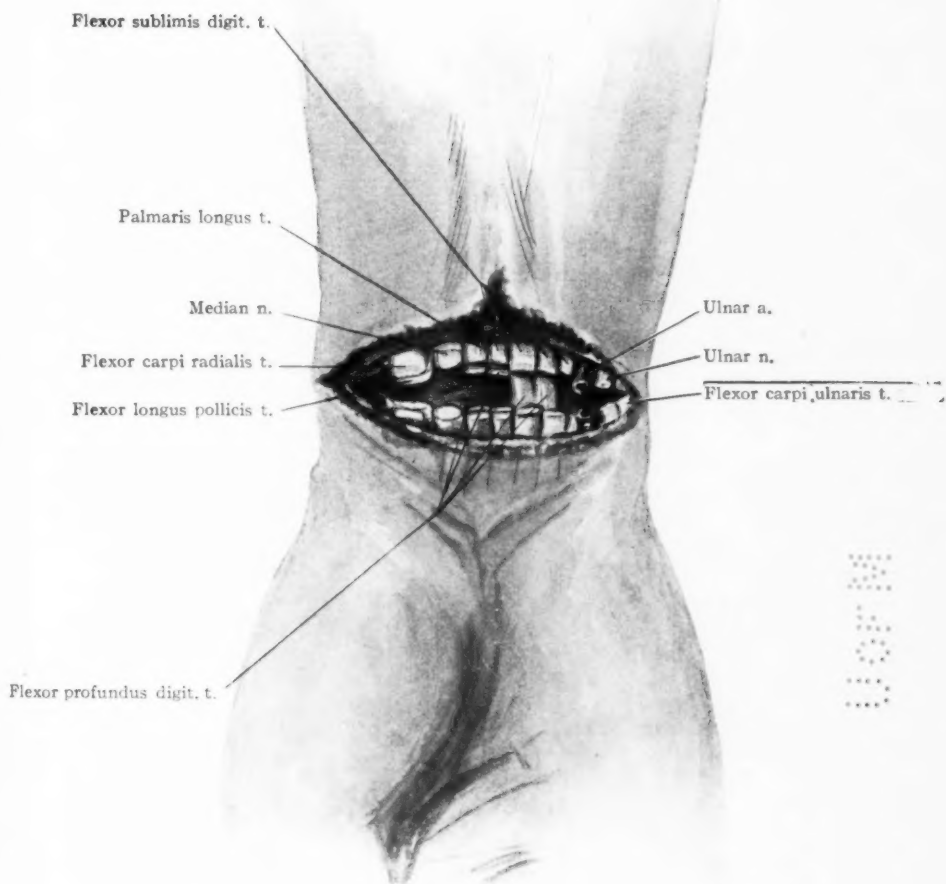
F. N., thirty-seven years of age, machinist. While at work the patient fell a distance of about 15 feet onto a skylight, the latter breaking, and he received an incised wound of the right wrist in addition to other injuries. Admitted to St. Mary's Hospital August 3, 1910. Treated by Dr. Wolf, Resident Physician. Ether anæsthesia.

Examination on admission showed a transverse lacerated wound about two inches in length of the flexor surface of the right forearm about one inch above the hand. Separation of the edges of the wound showed complete division of the flexor carpi ulnaris, the palmaris longus, the flexor carpi radialis, the flexor longus pollicis, the flexor sublimis digitorum, the flexor profundus digitorum excepting the divisions to the ring and little fingers of the latter; division of the median and ulnar nerves; and of the ulnar artery.

The forearm was thoroughly cleansed with soap and sterile water and alcohol 70 per cent. The wound was then irrigated with hot normal salt solution. The wound was then enlarged; the divided tendons were united separately with interrupted sutures of No. 1 chromicized catgut; the ulnar artery was ligated, and the divided ends of the median and ulnar nerves were separately united with through-and-through interrupted sutures of No. 1 chromicized catgut. The wound was then closed by interrupted sutures of silkworm gut. A dry sterile dressing was applied, and the hand and fingers placed in a position of marked flexion. The wound did nicely until the fourth or fifth day, when it was necessary to remove several sutures on the radial side of the wound, and it was found that there was a moderate degree of suppuration present which eventually resulted in a partial sloughing of the tendon of the flexor longus pollicis and separation of the cut ends. At the end of three weeks the wound had entirely healed. The patient has had very thorough and efficient massage since leaving the hospital, and is now at work.

Examination March 30, 1911, showed very little atrophy of muscles, extension of the fingers was good, excepting that of the distal phalanges; extension of the thumb was limited by the fixation of the distal end of the flexor longus pollicis at the wrist, and flexion was absent for the same reason; and in addition it was apparent that the site of suture had given way, and the cut proximal end had retracted. Flexion of the fingers was good in all, but was still somewhat diminished in the ring and little fingers. Complete flexion was limited by adhesion of the tendons at the site of the injury. Trophic changes were still present, although rapidly diminishing on the fingers; the flexor surface of the fingers felt cold, also tips on extensor surfaces, skin was atrophic, and nails were slightly roughened and longitudinally striated.

FIG. 11.



Showing the incised wound and the involved structures. (Case V.)

FIG. 12.



Showing the degree of extension possible in the fingers, and the fixation of the thumb
(Case V.)

FIG. 13.



Showing the degree of flexion possible. (Case V.)

340

FIG. 12.



Showing the degree of extension possible in the fingers, and the fixation of the thumb
(Case V.)

FIG. 13.



Showing the degree of flexion possible. (Case V.)

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XXXXX
XXXXX
XXXXX
XXXXX
XXXXX
XXXXX
XXXXX
XXXXX
XXXXX

The most common cause of incised and lacerated wounds of the extremities is from machinery accidents, gunshot wounds, explosions, cuts from scythes, glass, and sharp cutting instruments. In these accidents there is not only division of the superficial structures, but generally also division of muscles, tendons, blood-vessels, and nerves. In addition to the injury of the special structures there is very frequently, in lacerated wounds, a considerable destruction of the skin.

On first examining these wounds many problems are to be considered, dependent upon the extent of the wound and the structures involved. In the severe lacerated and incised wounds, where formerly amputation was the justifiable procedure, to-day entire parts are saved, useful limbs are preserved, and good functional results obtained by suture of muscles, blood-vessels, nerves, and tendons. Very frequently considerable judgment is necessary to come to the best possible conclusion, and in some cases operative procedure should be delayed on account of the shock so frequently present in many of these cases. Our primary interference should be limited to moderate cleansing, the removal of imbedded portions of clothing and foreign bodies, the ligation of small arteries, and an antiseptic dressing applied to the parts until the patient has thoroughly recovered from shock.

One of the most important problems in the treatment of these wounds is that of infection introduced at the time of the accident, either from the patient's clothing, his skin, or from the foreign body that produced the wound. Another very important consideration is the period that has elapsed from the time in which the wound was received and the time of seeing the patient. These two points have a great determining factor in the production of infection. In the treatment of these wounds they must all be generally considered as infected. We, however, know that very many of them can be thoroughly cleansed, and that they will heal by primary union. This is especially so of incised wounds. It is only from experience that we learn which ones should be drained, and which will probably heal without drainage. The chief infections to be

feared are tetanus, streptococcus, gas bacillus, and staphylococcus forms. The great determining factors in the production of infection are the condition of the parts, clothing, and trauma at the time of the injury. A secondary determining factor in the extent of infection is the amount of the destruction of tissue produced by crushing. In addition to infection the most dangerous immediate effect of these wounds is hemorrhage. Fortunately to-day our means of combating hemorrhage are very efficient, and in the use of normal saline solution by hypodermoclysis, proctoclysis, intravenously, and in the more urgent cases by direct blood transfusion we are generally able to meet all of these cases successfully, if the patient is seen early enough and there is not too great a degree of shock present.

In many of these wounds the chances of primary union are not good unless they are caused by clean, sharp cutting instruments and thorough cleansing of the parts is instituted. In a large majority the edges of the wound are primarily grossly infected, crushed, and devitalized, and in spite of the most thorough cleansing, dirt and grease cannot be entirely removed from the skin, and the edges of the wound in its entire depth have been so badly devitalized that either sloughing occurs in clean wounds or the resistance of the parts has been so lowered that infection readily occurs. An interesting form of treatment in these cases is that suggested by Reclus, who prefers never to irrigate with antiseptic solutions, who does not advise primary closure of the wound, and who dresses the wound with antiseptic ointment of vaseline 300, antipyrine 5, boric acid 3, salol 3, iodoform 1, carbolic acid 1, bichloride of mercury 10. He irrigates the wound with hot water under high pressure and then places the above ointment directly to the wound. In the treatment of these wounds I think that the best results are to be obtained from the following methods: thorough washing with soap and water, shaving the entire part, a second washing with soap and water using a firm brush, then washing the parts with alcohol 70 per cent. for two minutes, and in cases where the skin is covered with grease or

any other oily substance to wash with ether. The wound should be thoroughly irrigated with hot normal saline solution, all foreign particles removed, and all badly soiled and devitalized tissue should be cut away with a sharp knife.

After the above cleansing has been performed, a careful examination of the wound should be made for divided muscles, tendons, blood-vessels and nerves. The thorough approximation of these structures, especially the last two, is very important. Very often it will be found necessary to enlarge the superficial wound and to make a rather extensive dissection before the divided ends of all of the cut structures can be found. When deliberate suture has been performed, drainage of the wound must depend upon the degree of soiling by infectious material, the length of time that has elapsed since the accident, the amount of pressure destruction of the edges of the wound, and its position. The importance of a careful search for cut structures cannot be too greatly emphasized. How frequently do we see cases of comparatively trivial superficial wounds, in which division of important tendons and nerves has not been recognized until the resulting paralysis and atrophy call our attention to the nature of the injury!

OBESITY AND ITS SURGICAL TREATMENT BY LIPECTOMY.*

BY H. EDWARD CASTLE, M.D.,
OF SAN FRANCISCO, CAL.

It is a well-known fact that fat when in excess fails to perform the function for which it is intended, and becomes a burden to the life of its possessor. Adipose tissue has for some of its functions the preservation of the body, and the formation of beauty by producing symmetrical outlines. It fails to protect its possessor when it accumulates to such a degree that he is unable to perform the duties of life without extra exertion. When the inactivities of age begin to come to a person, his sedentary habits are greatly enhanced if he is endowed with an excess of fat. Thus the vital organs concerned in the metabolic processes of the body are incapacitated, and as the intake of food often remains the same, the result is, unfortunately, an increase of body weight.

If the adiposity is generally distributed we can do nothing but properly regulate the diet, exercise, hygiene, and general physical condition of the patient. Such patients are, obviously, not in the realm of the surgeon. They are subjects of the internist and should receive his careful attention. These patients are not interesting; there is very little to stimulate a scientific interest; they need constant attention to prevent them from regressing into their former modes of living, thereby undoing all that has been accomplished; and they are often discouraged at their slow progress. The result of this unfortunate fate is they read of and hear of advertised remedies and men who can reduce their weight rapidly, and they become martyrs to injurious chemicals and are the subjects of extortion by fraudulent men.

There is another class of patients to treat who are suffering

* Read before the Society of the San Francisco Polyclinic and Post-Graduate School, August 2, 1911.

from excess of fat. In these there are large localized areas of fat that can be removed to the advantage of its possessor. After discarding lipomas there are still collections of adipose tissue that are very detrimental to the welfare of the one to whom such belong. This is strikingly realistic in the abdomen of many people. Persons so afflicted cannot take the proper exercise; gymnastics and walking fatigue them so rapidly they do not get enough of either to be of much avail. The excess of weight often breaks down the arches of the feet, which condition is frequently associated with varicose veins of the legs, and ulcers. Sedentary habits are thus enhanced with sequelæ of incompetent liver, constipation, myocardial changes, etc. The contour of the body is unsightly, which keeps the individual out of society, and, if it chances to be a woman, induces her to wear harmful corsets to mold the form into a presentable appearance. The great weight outside of the centre of gravity gives her a mien which is ungraceful and embarrassing. Eczema is prone to appear in the folds during hot weather.

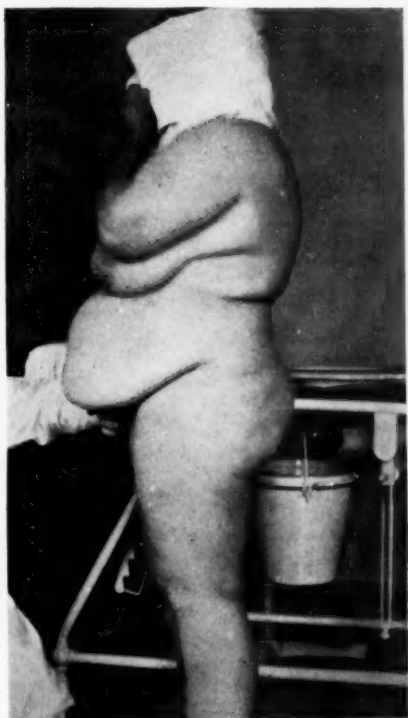
Some of these unfortunate individuals have been relieved by a surgical removal of the pendulous abdomen. There remains a large number of people so afflicted who would submit to extirpation of the excess of such circumscribed areas of fat if their surgeons would suggest it and advise them as to its ultimate benefits.

The operation is of the most simple form in surgery and consists of an elliptical incision which encompasses the amount of tissue to be removed, and which extends down to the deep fascia. The area thus marked out can quickly be shaved from the deep fascia. We find the most useful instrument for this purpose is a medium size amputating knife. After ligating all bleeding vessels, the superficial fascia should be closed with catgut sutures and the edges of the skin brought together by interrupted silkworm gut. These sutures should be placed about one inch apart and about one centimetre from the edge of the skin. Very accurate apposition of the skin is very desirable, and is best brought about by a continuous horse-hair suture carefully placed. The greater the heed to this detail

the more ready will be the healing of the wound. If due attention be paid to the closure of the wound, the scar will not be too unsightly and the convalescence will be much more agreeable to both patient and surgeon. The wound should be dressed with gauze which has been moistened with boric acid solution; this enhances the capillary attraction of the dressing and is a potential factor in relieving post-operative pain. If the binder which is destined to hold the dressings in place be drawn moderately snug the pain will be reduced to a minimum. It is easy to estimate the amount of tissue to be removed by endeavoring to approximate the fingers of the two hands when the mass is between them. If there is any tension on the sutures it can be relieved by flexing the body by the aid of a back rest in the bed. Local anæsthesia is not preferable in these cases, as the incision is too great in length. It is better to use general anæsthesia, the choice being nitrous oxide and oxygen, if there are no contraindications to its employment.

The operation was first called to the attention of the profession in 1890 by Demars and Marx.¹ Professor Howard A. Kelly² reported a case in 1899. The work of these gentlemen did not receive wide publicity, for we are unable to find many cases reported. Maylard³ in 1907, Schulz⁴ in 1908, and Weinhold⁵ in 1909, all published records of their cases. In 1910 Kelly⁶ wrote again advising the operation, giving it the name of lipectomy, describing this simple technic and reporting a very illustrative case. In the same year he pointed out the applicability of lipectomy while operating on very fleshy patients in order to make it possible for the operator to gain closer access to deep-seated pelvic lesions.⁷ Ballard⁸ reported one successful case in 1910 and Oehlecker⁹ in 1911; both performed lipectomy while operating for umbilical hernia. In the same year R. Jolly¹⁰ described his method of removing the excess of fat from the abdominal wall and reported two cases. It is possible that other cases have been recorded, but, if so, they are few, and we have not been able to learn of them during a careful perusal of the literature. Then, too, there are undoubtedly a large number which have not been

FIG. 1.



Shows pendulous abdomen.

FIG. 2.



Patient two months after operation.

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OF
THE
WORLD
BY
J. R. R. TOLKIEN

published. It is the object of the writer in reporting this case to help in keeping this valuable operation in the minds of the progressive surgeons until it receives its deserved attention.

CASE I.—Lady, thirty-eight years old. A native of Sweden. Had had diseases of childhood. Menstruated at twelve years, irregular, and accompanied by pain. Married ten years; husband has nephritis. Two children; one is well; one has a nervous disorder which causes him to have incontinence of fæces when at work in school. Two years ago patient had a miscarriage which took place in the third month of gestation. Since this sickness she menstruates for ten days each month, during which time there is profuse flow. Patient is very constipated. She becomes fatigued and out of breath when taking the slightest exercise.

Physical Examination.—Heart action very irregular; this irregularity is markedly increased during exercise. Patient is 5 feet $3\frac{1}{2}$ inches in height, and weighs 300 pounds. The adipose tissue is abundant in the abdomen and in the breasts, effecting a very awkward position while standing and extreme labor while walking. Both legs have large varicose veins below the knees; midway between the ankle and the knee on the anterior and the anterolateral surfaces of the right leg is a varicose ulcer which is 7 cm. in diameter. Trendelenburg's test for function of the valves in the internal saphenous veins shows they are competent.

The patient was sent to the hospital for Schede's varicose vein operation and Kelly's lipectomy.

Preparation of Patient.—Forty-eight hours prior to operation the ulcer and leg were cleansed with soap and water, then dressed with a gauze dressing which had been moistened in a solution of bichloride of mercury the strength of which was 1:10,000. The evening immediately preceding the morning of operation the abdomen and back were cleansed and covered with sterile, dry gauze.

Operation.—Under ether anæsthesia both legs were operated for varicose veins after Schede's method. The abdomen was painted with three coats of tincture of iodine. An incision beginning three inches laterally from the spinous process of the

first lumbar vertebra was made to traverse the anterior abdominal wall above the umbilicus, ending on the opposite side at a point analogous to the point of origin. The ends of this incision were joined by a second incision traversing the anterior abdominal wall a few inches above the pubis. The tissue composing the area thus outlined was removed from the deep fascia by the employment of a medium size amputating knife. The skin was closed as advised above. The line of suture was reinforced by silkworm tension sutures placed about three inches apart and including in their grasp the entire coats of the abdominal wall which were external to the deep fascia. These sutures were introduced one and one-half inches on either side of the suture line. Drainage was effected on either side of the body at the most dependent part, and the wound dressed with a large, moist boric acid dressing.

Subsequent History.—Schede operation healed without incident. The ulcer healed rapidly under scarlet-red ointment treatment.

During the three succeeding days following the operation the wound in the abdomen exuded a large amount of serum. Healing was *per primam*. The stitches were removed the ninth day after operation, and the wound supported by a collodion dressing. Patient walked out of the hospital the thirteenth day. During the time she was in bed she received no nourishment except water and three glasses of milk per day. Since her convalescence her diet has been carefully selected so that she has gained none of the 65 pounds which she lost during operation and starvation. She now claims to feel better when at work than she formerly did when at rest. The accompanying photographs depict her condition before and after operation.

BIBLIOGRAPHY.

- ¹ Demars and Marx: *Le progrès médical*, 5, iv, 1890.
- ² Kelly: *Proc. Johns Hopkins Bulletin*, vol. x, 1899, p. 197.
- ³ Maylard: *Brit. Med. Jour.*, Oct. 5, 1907, p. 895.
- ⁴ Schulz: *Mitt. a. d. Grenzgebieten d. Med. u. Chir.*, 1908, Bd. xviii, H. 5.
- ⁵ Weinhold: *Zentral. f. Gyn.*, Sept. 18, 1909, p. 1332.
- ⁶ Kelly: *Surg., Gyn., and Obst.*, March, 1910.
- ⁷ Kelly: *ANNALS OF SURG.*, March, 1911, p. 364.
- ⁸ Ballard: *Oklahoma State Jour.*, Nov., 1910.
- ⁹ Oehlecker: *Zentral. f. Chir.*, 1911, No. 11.
- ¹⁰ Jolly: *Berliner klinische Wochenschrift*, July 17, 1911.

BOOK REVIEWS.

VAGINAL CÆLIOTOMY. By S. WYLLIS BANDLER, M.D., Adjunct Professor of Diseases of Women, New York Post-Graduate Medical School and Hospital. Octavo of 450 pages, with 148 illustrations. Philadelphia and London, W. B. Saunders Company, 1911.

The contents of this treatise are divided as follows: posterior vaginal cœliotomy; anterior vaginal cœliotomy; simple vaginal hysterectomy; indications and limitations in disease of adnexa; hysterectomy for myomata, submucous, deep interstitial tumors, and intraligamentous growths, hysterectomy and technic with indications for vaginal Cæsarean section.

The distinctive character of the work is the technic of the several operations discussed.

Relatively speaking, the book is neither dogmatic nor controversial, but the author points out in terse, plain language the reasons for his beliefs and methods. A tone of unobtrusive confidence pervades the book and impresses the reader that its author is speaking logically and from an experience which lends authority to its teachings. Concise and positive statement without circumlocution has enabled the writer to cover his ground in a comparatively small volume, and this fact adds immensely to its intrinsic value.

From the author's standpoint, the student and the operator are not left in doubt as to the technic of any operation, a fact not always easy to comprehend from didactic teaching. The art of the illustrators is carried to a high degree of perfection, so ideal that the reader sometimes questions whether they obscure the real.

After reading the book one is impressed with the conscious confidence and finality of its teachings, and admiration for the elegance of its diction.

Probably the subject of posterior vaginal cœliotomy comes first in point of value and ability. The usefulness of this procedure is of the highest importance in the ascites of peritoneal tuberculosis, and within certain limitations, paradoxical as it may appear, makes this form of tuberculosis easily curable. Again

the author gives proper emphasis to its value in pelvic abscess. Whenever there may be uncertainty as to pus being in the peritoneal cavity or in the connective tissues lateral or posterior to the uterus, the procedure is equally wise and valuable.

Posterior cœliotomy for delivery of uterus and for removing ovarian cysts after Dührssen's method is warmly advocated.

While the author refers to Prior's most useful practice concerning this route in pelvic peritonitis, he dismisses it without further comment.

The subject of anterior vaginal cœliotomy is reviewed in quoting the views of German authorities, as Sanger, Zweifel, Dührssen, Kuster from 1888 to 1905, and in 1896 of Wertheim in devising vaginal fixation and shortening of the round ligaments.

He also quotes American authorities in the use of the anterior vaginal route, mentioning Boldt's operation for vaginal hysterectomy in 1887, and Goff's early adoption of this route for retroversion, retroflexion, and conservative operations on the adnexa, together with myomectomy for fibroids and operations for ectopic gestation.

He fortifies his claim to the superiority of anterior vaginal cœliotomy by Abel's acknowledgment that abdominal operations are easier than vaginal, who also states his early objections to the vaginal route were due to the fact that he did not understand its technic.

Notwithstanding his sincere, forceful advocacy of the advantages of anterior cœliotomy, he is equally positive in the reasons for which it is contraindicated. Among these are pregnancy, involvement of the appendix, of co-existing necessity of exploring the gall-bladder, large irregular fibroids, intraligamentous tumors, hæmatomas high up, etc.

While it is impossible to go into detail of other operations recommended, that of vaginal Cæsarean section is entitled to more than passing notice. "We are able," he says, after Dührssen, "with the aid of vaginal Cæsarean section in all normal pelves, and in such pelves whose conjugate is not less than 7.5 or 8 cm., at any period of pregnancy or labor even without pains and a completely closed cervix, to empty the uterus of its contents within a few minutes." This is radical obstetrical surgery not likely to come into general use by the average obstetrician, but with a field of usefulness well established and likely to be enlarged in proportion as the confidence and technical ability of the accoucheur expand.

In no part of the splendidly illustrated treatise are the various types in operative procedure better shown than in that of the vaginal Cæsarean section, or their utility better enforced.

One point in the technic of most of these operations which challenges a reason for its adoption is the nearness to the external os—at which the operator begins his inverted double incisions.

Finally it may be stated that while the clearness and logic of the writer cannot be gainsaid, it is hardly probable that its teaching in the main will in any great extent influence many operators to abandon their preference for an abdominal cœliotomy.

WALTER B. CHASE.

DIAGNOSTIC AND THERAPEUTIC TECHNIC. By ALBERT S. MORROW, M.D., Adjunct Professor of Surgery in the New York Polyclinic. W. B. Saunders Co., Philadelphia and London, 1911.

The author has incorporated in this volume a large number of the procedures employed in the various methods of diagnosis and also of treatment. While some of the methods detailed belong essentially to the domain of the specialist, the majority are practical procedures which the general practitioner, and especially the hospital interne, may be called upon to perform at any time.

The plan of the work comprises, first, a description of certain general diagnostic and therapeutic methods and, second, a description of those measures employed in the diagnosis and treatment of diseases affecting special regions and organs of the body.

All procedures are given in detail, thus obviating the necessity of referring back to other portions of the text; this, however, causes unavoidable repetition which does not in any way detract from the value of the book.

The illustrations are well drawn and profuse. The present work deserves especial recommendation for the general knowledge which it conveys.

INDUCED CELL-REPRODUCTION AND CANCER. By HUGH CAMPBELL ROSS, M.R.C.S., L.R.C.P., with the assistance of JOHN WESTLEY CROPPER, M.B., M.Sc., M.R.C.S., L.R.C.P. P. Blakiston's Son and Co., Philadelphia, 1911.

This is a study in the isolation of the chemical causes of normal and augmented, asymmetrical human cell-division. It is a fascinating story of research along new lines, dealing with the microscopic study of living cells. Incidentally one

gets a view of the discouragement which British officials in India and Egypt throw in the way of the medical officers, to prevent their carrying on serious medical work. Dr. Ross has the courage to specify the Director-General of the Public Health Department of Egypt as such an official. This gentleman objected to Dr. Ross doing scientific work during his spare time, and also prevented his continuing his mosquito campaigns because he apparently did not believe in them; nor was Dr. Ross permitted to publish the results of his work. He left the Egypt service and continued his studies at the Royal Southern Hospital in Liverpool. His early discouragements at the hands of officialdom were not unlike those of his distinguished brother, Ronald Ross.

The investigators studied living cells in nutrient media under the microscope, and observed the means by which they could be made to divide and multiply at the will of the observer. With living cells in agar, they applied stains and various other substances, and observed their effect. As soon as a stain reached the nucleus of a cell it died.

Cells absorb all absorbable substances; they do not seem to have a selective power; they take out of the surrounding medium whatever is there, whether it is poisonous to them or not. Against this it may be offered that in therapeutics we observe that certain cells respond to certain substances and others do not. Ross and Cropper show that strychnine, for example, stimulates certain cells of the nervous system; but it is absorbed by all cells. It causes amœboid movements in leucocytes, but this gives rise to no gross symptom and passes unnoticed. A cell may absorb two substances, one of which is fatal to it and the other of which stimulates cell-division: it starts to divide and perishes in the process.

Experiments were made upon living blood-cells with aniline products, alkaloids, ptomaines, nucleins and glandular extracts. The proliferation of leucocytes and lymphocytes as a process of wound healing, it is shown, is caused by kreatin and xanthin. It has been supposed that the cell-proliferation of healing was due to the inherent propensity on the part of the cells to divide; but these observers have made it clear that division occurs when the cell has absorbed a definite amount of a chemical agent. Two of these agents are kreatin and xanthin, both of which are products of dead tissue, liberated by cells which have perished. When death

of cells takes place, then the neighboring cells cannot resist the absorption of these substances, and they proliferate.

Another substance which stimulates proliferation is globin, a histone derived from hæmoglobin. Hæmoglobin, it is shown, has a distinct action in causing proliferation of the malarial parasite. The most rapidly growing cancer is melanotic sarcoma; and the development of sarcoma in the presence of blood-clot or destroyed cells is well known.

Concerning leucocytes, the authors say that for nearly a century and a half these cells have been observed in the blood, but no one has heretofore seen them in process of division. Now, if they are made to absorb certain chemical agents, they divide immediately; and, what is more, the rapidity of onset and the time occupied by each division varies directly with the quantity of the substance absorbed. Cell-division seems to be a physical phenomenon which can be measured in the case of each cell in proportions of the amount of the chemical auxetics absorbed by them. It can be reduced to a mathematic equation. A wound produces the remains of dead tissues, containing kreatin and xanthin, and proliferation of leucocytes occurs in the presence of these agents. The cell-proliferation in healing is proportionate to the extent of cellular death: much destruction, much repair.

These investigators go on to show this common chemical cause of cell-proliferation. The spermatozoa furnish the extractives which start proliferation in the ovum. Globin applied to chronic ulcers stimulates cell-proliferation greatly. An alkaloid of putrefaction, like choline, it is shown, must be the cause of lymphadenoma. In connection with this latter statement, it is interesting to note that Trousseau, in 1872, stated that lymphadenoma often follows on a focus of suppuration.

In experimenting with epithelial cells, it was found that they could not be made to live long *in vitro*, and experiments were difficult. It was assumed that, as cancer is peculiar to that period of life when the dead cells in the body increase, it is a phenomenon similar to those already discussed. Normal blood-serum was found to contain a body which has a restraining effect upon all division induced by auxetics. It was found that 1 c.c. of serum contained in 10 c.c. of gelatin, which also contained 1 c.c. of a 1 per cent. solution of choline, stopped the kinetic action of these in exciting amœboid movements. The experimenters tried to increase the amount of this inhibiting body in cancer patients by injecting an augmented auxetic combined with blood-serum. Six

ounces of defibrinated sheep's blood were injected per rectum once daily. The serum contains the restraining body; and it was assumed that the red cells would be destroyed in the rectum, the hæmoglobin decomposed and the globin augmented by the action of bacteria. The products in this combination, being absorbed, it was thought, might act as a sort of vaccine.

This treatment the authors report having applied to several cases with good results in most of them. In the cases most carefully studied the tumor was made to disappear. The criticism should be offered that more cases should have been studied before publishing this report. It is of value in throwing light upon the cause of cell-proliferation. To these authors, perhaps, belongs the credit of having shown the cause of normal human cell-division, and possibly the cause of malignant cell-proliferation also. Whether the therapeutic measures which are suggested shall be of value remains to be seen.

JAMES P. WARBASSE.

PRACTICAL CYSTOSCOPY. By PAUL M. PILCHER, A.M., M.D., Brooklyn, N. Y. 8vo, pp. 398. W. B. Saunders Co., Philadelphia and London.

A work on cystoscopy in English has been much needed. Dr. Pilcher's book supplies this need in a most satisfactory manner. The arrangement of the work is excellent, the text clear and concise, the illustrations, with liberal descriptive legend, are unusually instructive, and the work as a piece of bookmaking reflects credit upon the publishers.

The subject matter is divided into seven parts.

Part I deals with the types and construction of the various cystoscopes now in use, the electrical devices for supplying illumination, the technic of conducting the examination both in the male and female. The diagrams and illustrations to aid in interpreting objects seen through the different types of cystoscopes are excellent. There are many practical suggestions in regard to conducting the examination which are of much value to beginners. The technic of ureter catheterization is clearly described and illustrated. The difficulties encountered and the means at our command for overcoming these difficulties are well put.

Part II deals with the pathological conditions of the bladder as seen by the cystoscope. The various diseases are sufficiently described, and with the numerous excellent illustrations accompanying the description the different diseases should be recog-

nized, one from the other, with less practical experience than was heretofore necessary.

Part III deals with diseases of the prostate. Here the method of recognizing the different diseases of this organ and the special forms of prostatic hypertrophy are described and illustrated. Diseases simulating prostatic diseases symptomatically are mentioned and their differential characteristics noted.

Part IV deals with diseases of the ureters. The methods of recognizing the diseases of these structures by means of the ureter catheters and the Röntgen rays are considered. The important information to be derived from distending the ureters with solutions of silver salt in connection with Röntgen ray examination might perhaps have received more consideration.

Part V is devoted to the consideration of the methods of determining the functional activity of the kidneys. The different tests are described and their relative value discussed. The exact details of technic are insufficient for those with no knowledge of the subject, and the student must consult original articles or other sources for this detail instruction, some of which original articles are designated by footnote.

Part VI considers the many different means of diagnosis of the various diseases of the kidney. Helpful illustrative cases are cited. The value and methods of local therapeutic measures are here considered. Semi-diagrammatic illustrations showing the pathological conditions and the appearances of ureteral orifices in the different diseases are most valuable.

Part VII describes briefly but sufficiently the important operative procedures which may be carried out by special cystoscopic instruments and devices.

Considering the work as a whole, Dr. Pilcher's book must be considered complete, well balanced in regard to the amount of space devoted to the different subjects, the arrangement excellent, and text and illustrations cannot be too highly commended. The index is most satisfactory. The book is typically American and has no superior in any language.

JOHN H. CUNNINGHAM, JR.

PLASTIC AND COSMETIC SURGERY. By FREDERICK STRANGE KOLLE, M.D. New York, D. Appleton and Company, 1911.

The historic references in this book are of much interest. The author falls into the vernacular of the past and exclaims, "With-

al, it is a noble, generous art, worthy of far more extensive use than it now enjoys."

In the pages of the book which are devoted to the author's special field, we find descriptions of plastic and cosmetic operations, which the surgeon may study, with satisfaction. The descriptions of the various incisions for plastic work are the best to be found in surgical literature. In all operations, the early removal of sutures is recommended in order to prevent scar. On the face the stitches are removed in from twenty-four to forty-eight hours.

The author's operation for reducing the size of the ear is ingenious. His operation for malposition of the auricle is effective.

The operations for hair-lip are fully described. Incisions to meet every possible condition are illustrated. The same is true of the operations for restoration of the lips.

The use of paraffin in prosthetic surgery is fully and well discussed in all its aspects. Twenty-two possible untoward results are described, and means for their prevention given. The technic of injections is minutely described. A complete classification of the conditions which may be remedied by paraffin prosthesis is presented, together with the technic for each operation. The large possibilities of this branch of surgery are made clear by this book, and its mastery made possible.

All of the methods of rhinoplasty, which are worthy of consideration are presented. The methods are well described and illustrated. Especial consideration is given to the securing of bony or cartilaginous supports for the soft plastic flaps. The methods suggested are most helpful. Cosmetic rhinoplasty is so well described that ethical surgery might hope to rescue this field from the quacks.

This book is of especial value to the surgeon who is skilled in the principles of his art. He can refer to it with satisfaction when seeking information on the plastic surgery of the head, face and neck. It is a useful work, and in its special field fills a place in surgical literature.

J. P. WARBASSE.

COLLECTED PAPERS BY THE STAFF OF ST. MARY'S HOSPITAL, MAYO CLINIC. Volumes I and II. Octavo. Volume I, pages 668; Volume II, pages 633. W. B. Saunders Company, Philadelphia, Pa., 1911.

These two fine volumes present the papers and reports made by the staff of St. Mary's Hospital, of Rochester, Minn., from

1905 to 1911. Naturally the great bulk of the volumes is from the pens of the two senior members of the staff, Drs. William J. and Charles H. Mayo. In Volume I, out of seventy-three papers, Dr. William J. is to be credited with fifteen papers, and Dr. Charles H., with twelve. In Volume II, of sixty-three papers, Dr. William J. is to be credited with twelve and Dr. Charles H. with seven. When one observes that Volume II is the output from this clinic of a single year, the growth in the activity of the work is especially noticeable.

These papers have been read before various medical societies and are brought together in their present form for convenience of reference. More than that, however, in their collected form they constitute a magnificent monument to the scope of the work carried on in the Mayo Clinic, and to the industry, breadth and progressiveness which characterize that clinic.

The volumes are published with full Indices, both of authors and of subjects, so that reference to their contents is greatly facilitated. They are abundantly illustrated.

In running through these volumes one cannot fail to feel a certain personal note significant of the relations of the directors of this clinic to their whole work. These volumes form the beginning of a personal surgery of the highest character and of the greatest value. It is to be hoped that for many years to come successive volumes of the same character will appear.

THE TREATMENT OF FRACTURES, With Notes Upon a Few Common Dislocations. By CHARLES LOCKE SCUDDER, M.D. Seventh Edition, Revised and Enlarged. Octavo. Pages 708. W. B. Saunders Company, Philadelphia, Pa., 1911.

The successive editions of this book during the past ten years have received notice from time to time in the *ANNALS OF SURGERY*. It has been very interesting to note the steady growth in the scope and quality of the book. Its dominant note still remains that of treatment, and the number and character of the illustrations still remain a most attractive and valuable characteristic. The author has a genius for illustration, so that in turning over the pages of the book one seems to be in the presence of the author, demonstrating the particular condition under consideration. In the present edition it is to be noted that new material has been added, especially upon fractures of the skull, fractures of the spine, fractures of the neck of the femur, and injuries to the lower end of the tibia. A chapter on

the operative treatment of fractures has also been added. The attitude toward the operative treatment of fractures by the author is a very properly conservative one, such as every surgeon believes in, "that operative treatment of fractures is desirable, provided the indications for operation are clear." "The majority of simple or closed fractures can be satisfactorily treated by non-operative methods. Each individual case should be judged on its own merits." "It must ever be kept in mind that a very definite indication for operation must be present before an individual case is submitted to the additional risk of incision and direct fixation." These ideas we think will meet with general acceptance among surgeons. In their application, however, naturally there will be differences due to the different experiences, opportunities, and aptitudes of different surgeons.

Dr. Scudder's book has grown until it is no longer merely a hand-book, but it has become a portly volume of reference and occupies a worthy place among the very best books in its special field. We have put it on our shelf by the side of Hamilton, Stimson, Gurlt, and Hoffa.

LEWIS S. PILCHER.

CORRESPONDENCE.

NEEDLES FOR ARTERIAL ANASTOMOSIS.

EDITOR, ANNALS OF SURGERY:

Will you be so good as to call attention to an error in printing in the article "The Technic of End-to-End Arterial Anastomosis?" On page 489, the line "needles, No. 6 Kirby sharps" should read: "needles, No. 16 Kirby sharps."

Yours truly,

Boston, Mass.

ALBERT EHRENFRIED.

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